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COMMUNICATIONS

FAMILIAL HYALINE DYSTROPHY IN THE FUNDUS OCULI OR DOYNE'S FAMILY HONEYCOMB "CHOROIDITIS"

BY

M. TREE

LATE HOUSE SURGEON, OXFORD EYE HOSPITAL

LONDON

THE object of this paper is to deal particularly with a familial affection of the central and paracentral areas of the fundus, characterised by hyaline infiltration of the retina, and known as Doyne's honeycomb "Choroiditis," or Doyne's family "Choroiditis." The appearance of hyaline exudates in relation to the choroid and retina, occurs during the progress of various pathological conditions in the eye. An imperfect appreciation of the specific characteristics of widely different clinical entities, has led to difficulty in recognising and differentiating the fundamentally separate aetiological types.

Hyaline degeneration is very common in the eye, and is a product of altered local metabolism. It occurs as a degenerative change in inflammatory exudates; as a result of chronic toxaemias; as a local effect of vascular sclerosis; as one of the manifestations of senile change; and as an example of premature senescence in

ocular tissues, induced by abiotrophies of congenital and familial origin.

It is hoped that the characteristics of the familial affection, seen by the writer at different stages of its development, and here more fully dealt with than heretofore, will help in its early recognition, and to distinguish it from other types, and also assist in the elucidation of the tangled causes of related conditions.

In 1876, Jonathan Hutchinson published a report on 10 cases of "Symmetrical central choroido-retinal disease occurring in senile persons." These cases, though under the care of Hutchinson, had been observed by Waren Tay, who had also contributed the case-notes. Three of these cases reported were sisters, aged 60, 50, and 40 years. The family history revealed that their father and one of his brothers also had bad sight, and other members had nervous defects. The rest of the cases reported were obviously an heterogeneous collection of different types, and included such conditions as:—

(1) Old standing choroiditis with discrete large round atrophic patches in the periphery of the fundi (case 6).

(2) Doubtful renal retinitis with albuminuria and oedema (case 8).

(3) Tropical infections with traumatic rupture of the choroid and vascular changes (case 9).

(4) Probable syphilitic infection (case 10).

All these cases have gone down in the literature as examples of "Tay's central senile guttate choroiditis."

Apart from the fact that the report by Hutchinson and Tay contains the earliest reference to this type of familial degeneration in three sisters, the inclusion of seven other cases of central fundus changes with widely different causes, at once emphasises the need for appreciation of all the factors involved. Not only does this imply an insistence on the important fact that pathological processes of most diverse origin produce an ophthalmoscopic picture of whitish spots in the fundus, but also a reiteration of the fact that a particular degeneration, namely hyaline change, may be the end result of different diseases. A perusal of many communications published subsequently to that of Hutchinson and Tay, at once makes it evident that the above statement on the importance of careful differential diagnosis is not excessively laboured.

The first definite dissociation of a familial type of "central choroiditis," as a distinct clinical entity, was due to the painstaking observations of R. W. Doyne.

Doyne's observations were communicated to the Ophthalmological Society of the United Kingdom, with the demonstration

of affected cases (1899-1910); and he showed further cases at the British Medical Association's meeting in July, 1914. In 1913, Treacher Collins had the opportunity of preparing sections, and studying the histology in one of Doyne's cases. [case (4)].

The following may be taken as a summary of Doyne's observations on the condition :—

It first appears in early adult life (in one case observed at 20 years of age), but much more commonly later (*i.e.*, at about 40 years of age). It may either affect the disc neighbourhood, the macular area, or the " disc-macula " area. It consists of circular whitish patches of exudation. These increase in size and number during middle age, and set up some irritation and pigmentary disturbance, for though gross pigmentation is not a usual appearance, in some cases there is a good deal to be seen. The spots of exudate become agglomerated in the " disc-macula " area, and present a closely grouped distinctive, honeycomb appearance. In old age the condition passes into atrophy, the discrete outlines of the spots forming the honeycomb disappear, merging to form a more or less homogeneously atrophic area. In early cases there is no disturbance of vision. Even when gross lesions are found, the vision does not appear to be proportionately affected. When the lesions become confluent and atrophy ensues, there is a steady failure of sight. Both eyes are always involved, and the condition is steadily though very slowly progressive. It is hereditary, affecing several members of the same family in different generations. No special family diathesis can be detected.

Treacher Collins's report on the eye submitted to section, was in substance, as follows :—

A new formation of hyaline substance is found situated between the retina and choroid, the inner surface of which presents nodular elevations, with depressions between them. In places the hyaline material is covered by a single layer of pigment epithelial cells, in others it is devoid of any such covering, whilst here and there in the recesses between the nodules, heaped up masses of pigment cells are seen. The only changes to be found in the retina and choroid are such as can be accounted for by the pressure of the hyaline mass, which is evidently of primary formation, and attributable to some hereditary degeneration in the pigment epithelium.

At this stage it is useful to recall the normal histology of the parts, and to recognise that the membrane of Bruch is normally a glass-like hyaline structure, and that it is upon the surface of the membrane of Bruch that these new exudates are formed. In considering the origin of the exudates, it is helpful to deal further with the formation of Bruch's membrane. This membrane when stained with Weigert's Elastin is seen to be composed of two

layers, an inner which is homogeneous and becomes diffusely blue, and an outer which is finely reticular, and takes on the deep colour characteristic of elastic tissue. The difference between the two layers shows best where the membrane is thickest, *i.e.*, in the vicinity of the optic disc. At the margin of the papilla they end at different levels, the inner one ceasing with the pigment epithelium, and the outer continuing on for some little distance into the substance of the papilla. These two layers have different origins, the inner homogeneous layer, like the capsule of the lens and Descemet's membrane, which it closely resembles, being formed as a kind of secretion from the pigment epithelial cells which line it; and the outer reticular elastic fibre layer being continuous with the elastic fibre network of the choroid. The inner homogeneous part of the membrane of Bruch, though it becomes slightly thickened with the advance of years, is mainly a product of the pigment epithelial cells during embryonic life. Under normal conditions the capacity which the cells have of producing hyaline tissue ceases in the fully developed eye. Under pathological conditions the pigment epithelium re-acquires this hyaline-producing capacity, and nodules of it may be formed on the inner surface of Bruch's membrane. While recent, small, hyaline nodules remain homogeneous, the larger and older ones may show lamination and calcareous granules in the centre.

In other cases similar hyaline nodules form in connection with the termination of the elastic lamina at the margin of the optic disc, giving rise to elevations on the margin. These latter have to be distinguished from the *Drusen* bodies arising from the centre of the disc, and are demarcated by their marginal site, and their continuity with the elastic lamina. The central *Drusen* bodies in the papilla are usually grouped around the retinal vessels in front of the lamina cribrosa, are not connected with the membrane of Bruch, and are presumably not produced by the pigment epithelium. It is possible that the central *Drusen* bodies are derived from the neuroglia of the optic nerve, for the cells of the neuroglia are derived from the pedicle of the developing optic vesicle, and the pigment epithelial cells from the outer layer of the vesicle. The presence of *Drusen* bodies with retinitis pigmentosa has been described, and it has been pointed out that overgrowth of neuroglia occurs in this latter condition. However, the aetiology is unsettled, and the following theories have been advanced. Lauber believed them to be due to misplaced pigment epithelium. Parsons believed they originated from degeneration following an earlier inflammatory process; while Fuchs favoured the origin from neuroglia. Their interest from the point of view of this paper lies in the fact that the large central *Drusen* bodies (which have been described in some cases as causing a swelling on the

disc of as much as 12 or 14 dioptres), have never been noted in any cases of Doyne's family " choroiditis."

Hyaline degenerations in other conditions are to be distinguished by the size, texture, situation, and arrangement of the exudates; and by a recognition of the characteristic progress, and associated clinical conditions. Thus the exudates in arterio-sclerotic retinitis are composed of hyaline spots in the outer molecular layer. The " cotton-wool patches " and the star figure of renal retinitis are similarly hyaline exudates, and stain strongly in the usual manner, with eosin, acid fuchsin, and picro-carmine; while they do not give the amyloid mahogany-brown reaction with iodine.

According to Leber the exudates in retinitis punctata albescens are also hyaline, while the same is probably true of retinitis circinata.

These related degenerations have here all been lightly touched upon, with the object of drawing attention to their associations. It is proposed to discuss them further after the main subject of this paper has been dealt with.

Reverting to the immediate details of the main subject matter, the writer collected the case notes of all likely patients who attended the Oxford Eye Hospital since 1896, and was able to find records of 25 cases. In six of these cases there was no record to be found of investigation of the family history, and therefore these are excluded. Of the 19 remaining cases a large number were dead, including the original case whose eye was submitted to section by Mr. Treacher Collins. The writer was, however, able to examine 7 of the 19 cases personally, on repeated occasions. During the process of examining other members of the families he was also able to discover one new unrecorded case [case (15)], and in addition 12 unaffected descendants were also examined. The records used thus consist of 20 affected cases and 12 unaffected descendants. Of the 20 affected cases—

9 can be grouped into one family tree [the High Wycombe family, cases (1)-(9)];

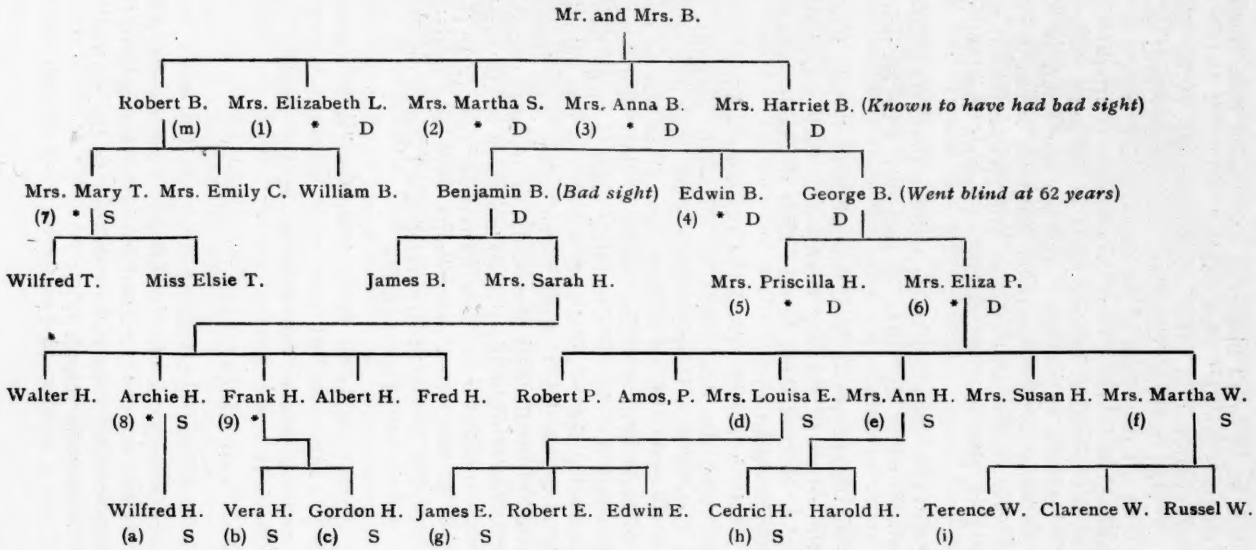
6 into another family tree [the Eynsham family, cases (10)-(15)];

2 other cases were sisters [cases (17) and (18)];

2 other cases were brothers [cases (19) and (20)], and

1 case [case (16)] gave a family history of six members with bad sight out of seven, and one sister was said to have been seen with the same condition as the patient, but no notes were found.

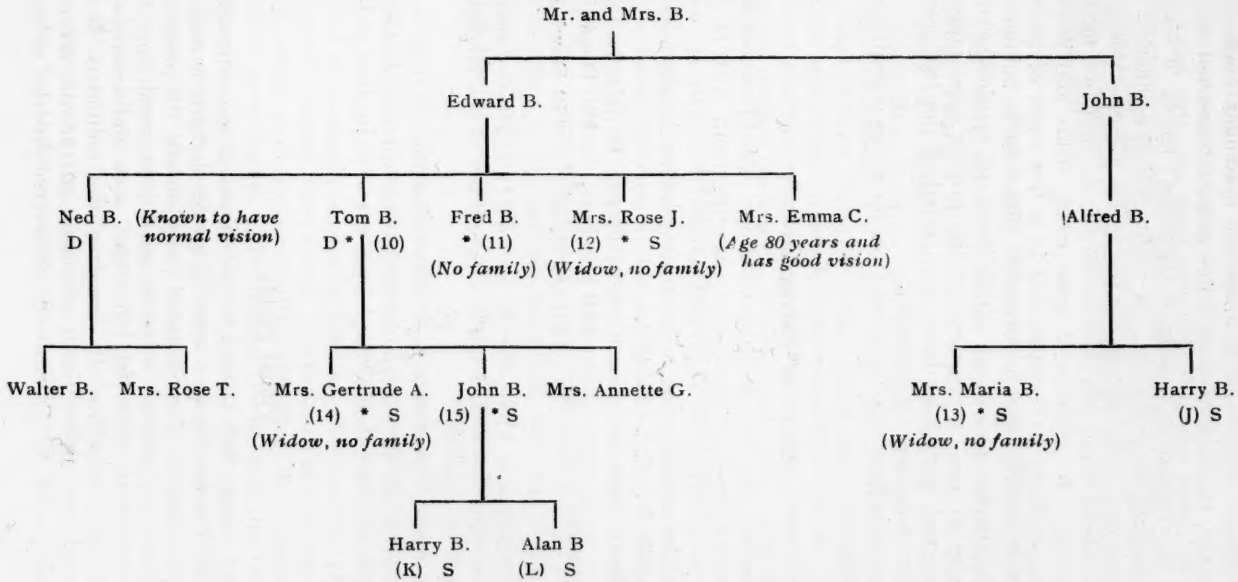
THE HIGH WYCOMBE FAMILY.



*—Affected cases examined at hospital.
S—Seen by writer.
(1), (2), etc.—Refer to case histories.
(a), (b), etc.—Case referei ce of unaffected descendants.
D—Known to be dead.

THE EYNSHAM FAMILY.

THE EYNSHAM FAMILY.



*—Affected cases examined at hospital.
S—Seen by writer.
(10), (11), etc.—Refer to case histories.
(J), (K), etc.—Case reference of unaffected descendants.
D—Known to be dead.

Of the 8 affected cases, and 12 unaffected descendants who the writer examined, the examinations where possible consisted of :—

A record of visual acuity, state of refraction; record of visual fields and charting of scotomata. A rapid clinical estimation of the light sense and colour thresholds (using Dr. George Young's album of threshold tests), and examination of the fundi and media under mydriatic. In addition in some cases, urine and blood-pressure examinations were made, and in a few cases of blood-sigma test for syphilis was performed. The records submitted also include fundus paintings, which serve to emphasise the various stages in the development of the fundus appearances. Two genealogical trees have been constructed of the two main families referred to, and are appended.

The general conclusions to be drawn from the series investigated are as follows :—

State of Refraction

(i) In affected cases the refractive condition varied from emmetropia to small degrees of ametropia, extending from +2·0 D. to −1·50 D. In addition, in one case myopia of −3·50 D. was present, and in another case myopic astigmatism in both eyes occurred [−6·50 D.cyl.↓, case (6)].

(ii) In the 12 unaffected descendants [cases (*a*) to (*l*)], one had myopia of extreme degree [Rt. $\frac{-13{\cdot}0 \text{ D.sph.}}{-3{\cdot}0 \text{ D.cyl.}\rightarrow}$ L. $\frac{-13{\cdot}0 \text{ D.sph.}}{-3{\cdot}50 \text{ D.cyl.}\rightarrow}$ case (*h*)].

Three had myopia of from −3·0 D. to −4·0 D.; two very small amounts of myopia −0·50 and −0·75 D.; and five of the younger members were emmetropic.

From this it may justifiably be deduced that the affected eyes showed no marked tendency to hypermetropia and under development, but that the eyes of unaffected members were inclined to be more myopic.

Visual Fields

In affected cases the visual fields remained uncontracted, even when the fundus changes were advanced, and the exudates had become confluent. The advanced cases showed the presence of an irregular, paracentral scotoma in the temporal part of the fields, continuous with the blind spot area and extending towards the fixation point. In one case the scotomata were crescent-shaped and concentrically disposed around the central area [case (9)]. In the case newly discovered by the writer,

although the fundi showed marked lesions, no scotomata were observed and the vision was 6/5. [case (15)].

From this one must conclude that the visual fields show no abnormality in early cases; that the peripheral fields remain full throughout, and that gross disturbance of the retinal function is only manifested in advanced cases by the appearance of a paracentral scotoma.

The Light Sense and Colour Thresholds

These were rapidly estimated for each eye separately using Dr. George Young's Album of Threshold tests, with the patients wearing their appropriate lens corrections for reading distance.

Using these tests on the early case discovered by the writer with 6/5 vision, both light sense and colour thresholds were normal in both eyes (1st class). In grossly affected cases, both light and colour thresholds were equally reduced. It therefore seems that the estimation of light sense and colour thresholds, as by the method described, yields no additional useful information.

General Systemic Examination

No other obvious defects or associated systemic disturbances were found common to this condition. In a few cases the blood-sigma test for syphilis was performed and found negative. Similarly blood pressure estimations and urine examinations revealed no abnormalities. No associated mental disturbances were noted in the patients examined, nor was there any family history of mental trouble. In this latter respect the cases differ from the three sisters described by Hutchinson and Tay.

Examination of Fundi and Media

Except for the occasional occurrence of the usual senile lens changes in a few of the more aged patients, no abnormalities were noted in the media of affected patients. No posterior polar or complicated cataracts were noted. No instance of vitreous disturbance was seen; and no constantly associated developmental ocular defects were found. The retinal vessels and the colour of the optic discs were normal for patients at that age.

The average fundus appearance of a typical fully developed honeycomb " choroiditis," consists in the presence of a horizontally disposed oval area of closely packed rounded exudates, enclosed above and below by the upper and lower temporal branches of the retinal vessels. Laterally the area extends from the margins of the optic disc towards the temporal side of the fundus for about five disc diameters, while the vertical extent is

about three disc diameters. The exudate are greyish-white or putty-coloured, and are for the most part of uniform size and rounded. The average diameter of the spots is about 2-3 times that of the main retinal vessels at the disc. At this stage the spots are discrete and surrounded by shadow-rings suggesting a honeycomb or circular mosaic appearance. A few round exudates

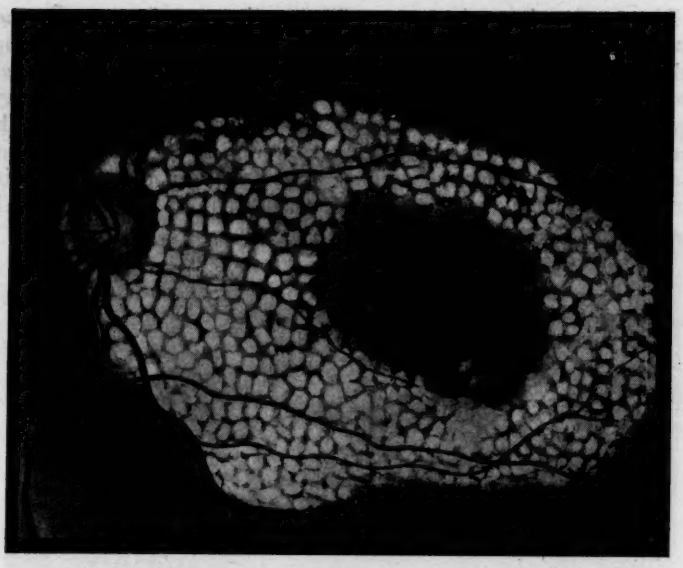
CASE 3.

are present on the disc margin deep to the retinal vessels, and a few isolated spots occur to the nasal side of the optic disc. A small amount of irregularly disposed pigment may be present, mostly in interlacing linear formation in the macular area, with some fainter cloudy pigmentation. As a temporary phenomenon a small amount of haemorrhage may be seen at the macular region, and this occurrence coincides with a decrease in visual acuity [cases (6) and (14)]. The whole condition is bilateral, though the appearances may vary in the two eyes and may be slightly more advanced in one than in the other.

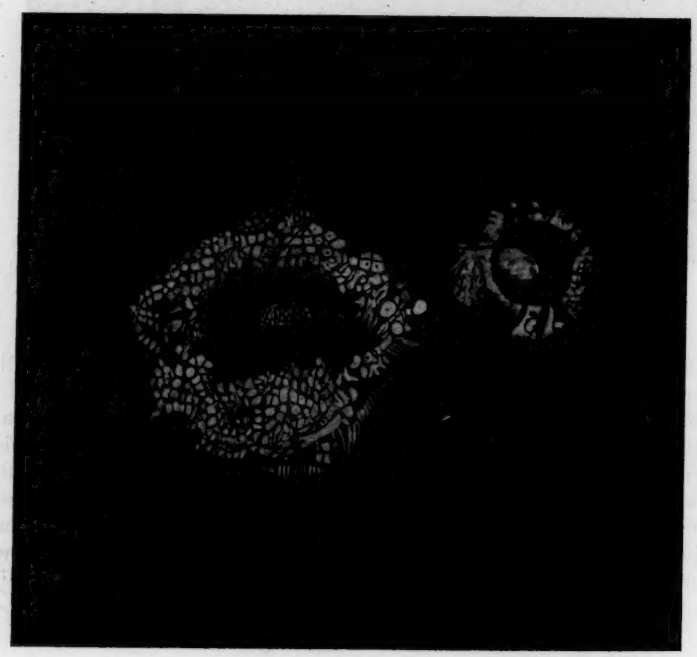
The fundus in early cases.—The earliest exudates are rather smaller than in the fully developed case, and are somewhat yellower in colour. The latter may presumably be due to their greater depth from the surface of the retina, and the accompanying diffusion of colour from adjacent vascular layers. The exudates occur first in the paramacular region especially between the disc and the macula. They spread in very close proximity around the macula and often encroach upon it at a very early

CASE 4.

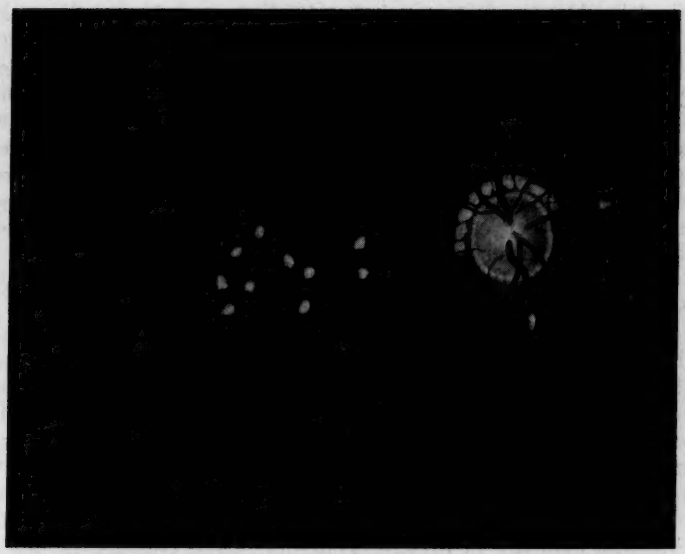
stage [case (15)]. In some cases the spots are disposed all round the macula in a ring [cases (3), (7), (10) and (11)], and the condition has then been likened to that in retinitis circinata. However, apart from the circinate disposure of exudates, there is little similarity between the conditions, as later discussion will show. Accompanying the para-macular exudates in early cases, there are also often a few spots resting on the margins of the optic discs, and occasionally a few tiny spots of exudate in the fundi to the nasal side of the discs. If any pigment is visible it is superficial to, and overlaps the exudates in the macular area.



CASE 6.



CASE 7.



CASE 15.

The fundus in advanced cases.—The advancement of fully developed cases, is characterised by the confluence of exudates, beginning in the centre of the affected area of the fundus, to form a pale yellowish plaque [cases (8), (12), (14)]. Before this process is completed the more peripheral discrete exudates are seen encircling the confluent zone. With further extension the plaque of exudate shows a definite raised margin delimiting the affected area [case (13)]. Meanwhile atrophic changes commence in the centre of the plaque, exposing the choroidal vessels to view [case (4)]. Extensive central atrophy may occur while the more peripheral encircling exudates still remain discrete. Such an appearance will give rise to yet another type of circinate phenomenon [case (4)].

Differential Diagnosis

An earlier reference has been made in this paper to the three sisters described by Hutchinson and Tay. The description given of these three cases conforms to that associated with Doyne's family "choroiditis," with the exception that there was also a history of serious mental defect in two other members of the family. Other cases without the element of family mental defect have since been described. Nettleship, in discussing some heriditary diseases of the eye (1906), referred to the condition des-

cribed by Doyne, and stated that he had seen several examples of " Tay's choroiditis " and sometimes more or less characteristic retinitis circinata,—in brothers or sisters. He described a man aged 72 years seen in 1896 with bilateral retinitis circinata. One of his sisters showed " a mixture of Tay's choroiditis and epithelial denudation." Another sister was said to have partly lost her sight from " disease at the back," while the mother of these three became blind as an old woman, " not from cataract." Nettleship described another case in an affected family where there were Tay's dots of larger size than usual.

In December, 1909, during a discussion initiated by R. W. Doyne, Harrison Butler described two cases he had seen in a mother and son. In May, 1910, G. T. Mould described a case where three members of the family were known to be affected. These additional cases, above referred to, serve to accentuate features of the condition previously described, and point especially to the necessity for differential diagnosis from retinitis circinata, and the non-familial type of Tay's choroiditis.

The non-familial type of Tay's choroiditis.—This condition presents minute pale yellowish spots, which are always small, with little evidence of fusion, occurring in old persons, as one of the manifestations of senility, and without any particular family proclivity.

Retinitis circinata.—Typical cases of retinitis circinata occur in elderly women with degeneration of the retinal vessels. The condition is associated with and follows retinal haemorrhage, and is unilateral in 50 per cent. of cases. Recovery with absorption of exudates is also known to occur. The appearance is that of a girdle of bright white patches of irregular shape and size, becoming confluent to form map-like figures, and disposed in a wide ring around the macular area. The latter shows yellowish-white areas, slight pigmentation, and often haemorrhages. In such cases, therefore, the girdle of exudates is whiter, larger, less regular, and more readily confluent, than in the family condition under consideration. The white exudates are limited to the girdle area, and in addition, no typical exudates occur on the margins of the optic disc. Further, the retinal vessels are diseased, both eyes are not always affected, the patient is old, and there is no family history.

As previously described, two stages in the evolution of Doyne's " choroiditis " may present a circinata appearance. In the early stages, the exudates may for a time be disposed around the macula in the form of a ring; but of course such exudates are discrete, round, and regular, and are disposed in more than one row, like a portion of a honeycomb. In the very late stage associated with atrophy, there will be a prolonged history of progressive loss of

vision, the affected eye will show exposure of the choroidal vessels in the macular area, and the other eye will be extensively involved.

Apart from retinitis circinata and Tay's choroiditis, perusal of the literature on this subject reveals that the following additional conditions have sometimes given rise to confusion, and therefore must be briefly considered:—

1. Cerebro-macular degenerations.
2. Retinitis punctata albescens.
3. Early mild disseminated choroiditis.
4. Arteriosclerotic retinitis.
5. Renal retinitis.

While late cases may also have to be distinguished from:—

6. Diffuse choroiditis; and
7. Exudative retinitis. (Coats).

Cerebro-macular degeneration may for convenience be considered in association with other heterogeneous cases, some of which may possibly be grouped with Batten-Mayou disease, and congenital day blindness. The exudates are usually limited to the macular area, the condition is noted at an earlier age, and mental defect in the patient is a prominent feature.

Retinitis punctata albescens presents the clinical signs of retinitis pigmentosa in addition to the presence of exudates. There is therefore night blindness with contracted visual fields or broad ring scotoma. The exudates are uniformly distributed over the entire fundus, with the exception of the immediate macular area. They are white and very small. Their average diameter is about half that of a medium sized retinal artery. In some of these cases, symptoms of cerebral degeneration also occur. In addition, some cases are seen intermediate in character between cerebro-macular degeneration and retinitis punctata albescens. Thus Nettleship refers to a case reported by R. D. Batten and E. H. Holthouse in 1897. The patient was a woman aged 25 years, with abundant choroidal exudates. She was the youngest of a family of 24, of whom 19 died early of some obscure brain disease. Dr. Guthrie who examined the patient believed her to be epileptic, and suggested that the condition was akin to retinitis pigmentosa. In addition, examination of the plate accompanying the case report, showed a different picture from that given by Doyne's family choroiditis. The exudates were mostly irregular in shape and this applied even to isolated discrete spots. Further the exudates were more widely distributed into the peripheral parts of the fundus.

Early mild disseminated choroiditis may involve the central part of the fundus. During the active stage, the edges of the exudates are hazy and not sharply defined; there are dust-like opacities in the vitreous, and precipitates may be seen on the

posterior surface of the cornea. The Wassermann reaction (W.R.) is often positive. These additional features, and the subsequent progress in affected areas, should make differential diagnosis fairly easy. In spite of which, this condition was obviously present among the cases originally described by Hutchinson and Tay.

Arteriosclerotic and renal retinitis may here be considered together. Case 8 of Hutchinson and Tay's series was probably of renal origin. The presence of cotton-wool patches and star figure at the macula, would of course easily distinguish the atypical nature of the condition. The small round exudates present in both renal and arteriosclerotic cases, are usually whiter and smaller than in the cases under consideration. In addition, the vascular changes, the possible unilateral distribution in arteriosclerotic cases, and albuminuria in renal cases, should facilitate differential diagnosis.

Diffuse choroiditis during the stage of exudation may here have to be distinguished from the advanced cases of Doyne's condition showing confluent exudates. However, in the former condition, as in true choroiditis, the exudate has no definite edge but shades off into the normal fundus; the affected area is irregular, and may finally involve the greater part of the fundus.

Exudative retinitis (*Coats*) may have to be considered especially in elderly patients where the affection is bilateral, or where the other eye shows the presence of retinitis circinata. It is possible that the latter condition is merely a less severe form of exudative retinitis, and both are probably due to retinal haemorrhage. Disease of the retinal vessels is a prominent feature, and there is a history of sudden onset of visual defect associated with retinal haemorrhage. The exudation may be typically massive, in which case the extensive continuous sheet of exudate covers the whole macular region. There is variation from place to place in colour, depth, and texture. No isolated discrete exudates will be seen along the margins; and if the second eye is involved there is not necessarily any relation between the extent of the lesions in both.

It is hoped that the preceding consideration of differential diagnosis will be helpful in eliminating doubtful cases. The appreciation of the stages of development of the fundus condition may be aided by reference to the accompanying fundus paintings. The case notes of the affected patients, and of unaffected descendants who were examined, are appended together with charts of visual fields and the genealogical trees of the two main families.

In conclusion I have pleasure in acknowledging my indebtedness to Mr. P. H. Adams for permission to use the records of cases at the Oxford Eye Hospital; and to Mr. A. Sorsby who suggested that these investigations might be usefully undertaken.

CASE 1. Mrs. ELIZABETH L.

Seen *March* 13, 1901, aged 52 years, with defective vision.

R.v. = 6/36 not improved. } Pupils equal and react to light and accommo-
L.v. = 6/60 not improved. } dation.

Condition of Fundi.—Both fundi show much the same appearance, but with more marked change in the right. Starting from the temporal side of the optic disc and extending somewhat beyond the macula, and vertically above and below the macula, is an area formed by closely aggregated circular white spots (like circular mosaic). There is a little pigment here and there, especially at the macula and around the optic disc—but speaking broadly, pigment is remarkable by its absence. The nasal side of the retina and the periphery generally seem normal. The pigment where it is seen is well forward in the retina, in some places masking the vessels.

March 27, 1901. The fine pigment patches seem to be far in front of the choroid, with evident parallax between the two.

March 26, 1902. R.v. = 6/60 } Fundi unchanged.
L.v. = 6/60 }

July 16, 1903. *In status quo.*

July 13, 1904, and *July* 23, 1906. Given Mist. pot. iodide.

CASE 2. Mrs. MARTHA S.

June 14, 1899, aged 53 years.

R.v. = 6/36 with +2·0 D. sph. = 6/12.

L.v. = 6/60 with +2·0 D. sph. = 6/12.

R. fundus.—Large pigmented crescentic patch at disc outwards numerous patches of choroidal exudate and black pigment in macular region and also down and out.

L. fundus.—Patch of black pigment at disc outwards. The macula normal but there are small white patches of choroidal exudate, scattered irregularly around the macu a, with fine lines of pigment.

July 13, 1904. R.v. 6/60 with +2·0 D. sph. = 6/24 one letter.
L.v. 6/60 with +2·0 D. sph. = 6/36.

Refraction under H. and C. +2·0 — +2·50 R. and L.

CASE 3 *with fundus painting.* Mrs. ANNA B.

March 27, 1901, aged 60 years. Complains of failing sight two years, especially during the last year.

Pupils equal and react.

R.v. = 6/60 with +1·0 D. sph. = 6/24.

L.v. = 6/36 with +1·50 D. sph. = 6/36.

Fundi.—Honeycomb choroiditis.

Ordered Mist. pot. iodide.

January 22, 1902. Rep. Mist.

Note added by the writer on appearance of fundus painting of R. eye.—The optic disc shows slight abnormality in its formation, below and to the nasal side. There is an irregular area of closely aggregated whitish exudates disposed around the macular region. The area of exudates is enclosed between the temporal branches of the retinal vessels. The immediate macular region is free of exudates. There is a small amount of fine cloudy pigment scattered around.

CASE 4 *with fundus painting.* Mr. EDWIN B.

February 9, 1910, aged 80 years. Complaining of bad vision for over 30 years and ? unable to read for over 20 years.

Vision both eyes = Counts fingers at 6 inches.

His mother had bad sight, and his two brothers were similarly affected.

Fundi.—The disc and macular areas are affected and show some scattered pigment. The affected area in each case is sharply defined from the surrounding normal choroid. In the area extending below the disc it is typically honeycomb.

Note added by writer on the appearance of fundus painting of R. eye—The condition portrayed shows advanced change. There is central atrophy with absorption of exudate and exposure of choroidal vessels. On the temporal side there is still a band of raised confluent exudate with a definite margin. Above and

below in the course of the temporal vessels, discrete round exudate are still present around the margin of the affected area. Below and to the nasal side of the disc there is a larger patch of agglomerated discrete exudates.

Case (4) provided the eye for section and histological examination by Mr. Treacher Collins in 1913, which was the date of this patient's death at the age of 83 years.

CASE 5. Mrs. PRISCILLA H.

July 21, 1904, aged 47 years.

? Guttate choroiditis present (no further notes on this date).

April 14, 1909. Honeycomb choroiditis.

R.v. 6/60 with −3·50 D. sph. = 6/24 one letter.

L.v. 6/60 no improvement.

July 4, 1910. Vision 4/60 R. and L.

CASE 6 *with fundus painting.* Mrs. ELIZA P.

July 21, 1904, aged 49 years. Complains of gradual failure of sight for 9 years.

R.v. $= \frac{6}{60}$ with $\frac{-0{\cdot}50 \text{ D. sph.}}{-6{\cdot}50 \text{ D. cyl.} \downarrow} = \frac{6}{60}$

L.v. $= \frac{6}{60}$ with $-6{\cdot}50$ D. cyl. $\downarrow = \frac{6}{18}$ one letter.

Visual fields fairly full.

Fundi show honeycomb choroiditis.

Writer's note on appearance of fundus painting of L. eye.—There is an extensive oval area of closely packed discrete exudates, extending laterally from the margins of the optic discs. There is a line of raised exudates along the margin of the temporal half of the optic disc. The macular region and an adjacent area together totalling more than two discs in size are free from exudates, but show the presence of haemorrhage and much local pigmentation.

CASE 7 *with fundus painting.* Mrs. MARY T.

November 19, 1913, aged 54 years, desires glasses for close work.

R.v. = 6/12. Pupils equal and active.

L.v. = 6/60 with −1.0 D. sph. = 6/18.

Fundi.—Honeycomb choroiditis in both eyes, more advanced in the *left*, where the macular region is more markedly involved.

Painting made of R. fundus.

September 2, 1914. R.v. = 6/12 one letter.
L.v. = 6/60.

Writer's note on appearance of fundus painting of R. eye.—The exudates are discrete and are mostly disposed in a small ring around and close to the macular area. The rows of exudates in the lower half of the ring, cover a wider area than those above. Some of the exudates are more clearly demarcated by pigment disposed around their margins. There is a larger irregular bunch of pigment close to the macula. Exudates are also present all round the margin of the optic disc.

Notes on writer's own examination.

December 10, 1935. (Patient is now 76 years.)

R.v. = 6/60 prefers +1·0 D. sph. but there is little improvement.

L.v. = counts fingers at 1 metre—not improved.

R. with +4·0 D. sph. = J.14. L. with +1·50 D. sph. = J.16.

Very early lens opacities in both eyes.

Fundi.—There is much exudate present in both fundi with branching and coarse pigment scattered around the disc and macular areas. There are yellowish exudates on the disc margins, but practically no choroidal change on the nasal side of the discs.

Blood pressure 162 mm. Hg systolic.

Urine, acid, S.G. 1020, no sugar or albumen.

January 8, 1936. Visual fields taken. Fields full.

Does not appreciate blind-spot areas.

CASE 8. Mr. ARCHIE H.

July 4, 1928, aged 45 years, Baker. Noticed slight defect in vision for 10 years.

R.v. = 6/12. L.v. = 6/18.

Fundi.—R. eye. Large area at macular region forming honeycomb type of choroiditis with some horizontal streaks of pigmentation. Similar collection of

circular white areas on upper edge of disc and some areas scattered about on the nasal side. *L. eye* very similar area. Spots above the disc and down and in from it with large area over macular region, and fine lines of pigmentation.

Notes on writer's examination.

March 1, 1936. R.v. $= \frac{6}{12}$ part with $\frac{-0{\cdot}50 \text{ D. sph.}}{-0{\cdot}75 \text{ D. cyl.} \downarrow 105°} = \frac{6}{9}$ part.

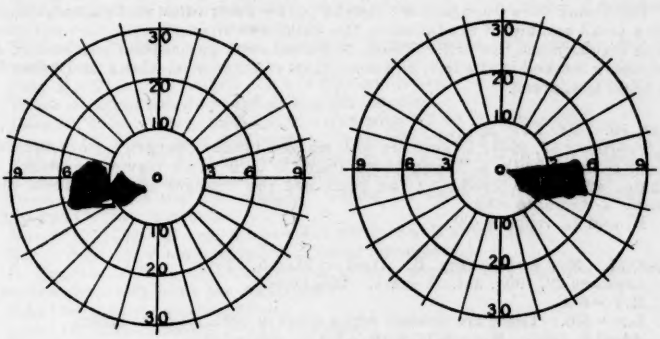
L.v. = 6/24.

L. with $\frac{-1{\cdot}50 \text{ D. sph.}}{+0{\cdot}75 \text{ D. cyl.} \rightarrow 180°} = \frac{6}{12}$ part.

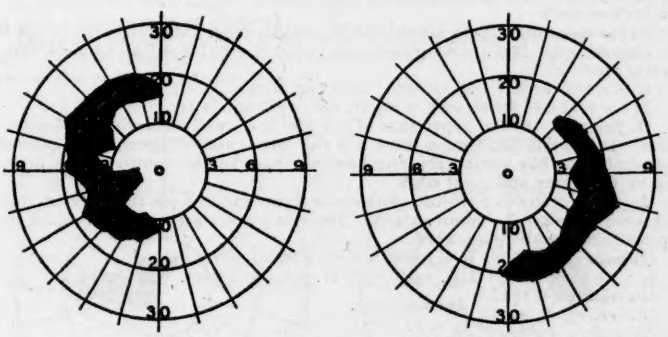
with added +1·50 D. sph. R.v. = J.8. L.v. = J.10

Fundi.—Very typical honeycomb exudates in both, with some exudates on nasal side of discs. The exudates are becoming confluent in the central region. There is very little fine linear pigmentation present.

The fields are full. There is a small *paracentral scotoma* in each visual field continuous with the blind spot and extending towards, but not quite up to, the central fixation point.



CASE 8. ARCHIE H. 1° White. March 1, 1936.



CASE 9. FRANK H. 1° White. March 1, 1936.

CASE 9. Mr. FRANK H.

February 27, 1907, aged 29 years, Chairmaker. Complains of failing vision for one year especially in right eye.

R.v. = 6/60 not improved. Pupils equal and active.

L.v. = 6/24.

R. fundus.—Large black patch of interlacing pigment at right macula with numerous white spots below, and some also under cover of the pigment.

L. fundus.—The left macula is similar to the right but shows much less pigment, and the white dots beneath are more visible, and more numerous around the pigment area. There are also a few on the nasal side of each disc. The vessels are fairly normal and run over the pigment.

Urine, nil abnormal found. *Ordered* Mist. pot. iodide.

July 26, 1907. Rep. Mist.

Notes on writer's examination.

March 1, 1936. Patient now 58 years.

R.v. $= \frac{6}{60}$ with difficulty with $\frac{-0{\cdot}25 \text{ D. sph.}}{+1{\cdot}50 \text{ D. cyl.}} \downarrow 65° = \frac{6}{60}$

L.v. $= \frac{6}{60}$ with $\frac{-1{\cdot}0 \text{ D. sph.}}{-1{\cdot}50 \text{ D. cyl.}} \downarrow 120° = \frac{6}{36}$

With addition of +2·0 D. sphs. = J.12.

There are early senile lens changes in both eyes especially in the right.

The fundi show changes very similar to the description of February 27, 1907, with a small amount of exudation on the nasal side of the discs.

The *visual fields* are full. There is a crescentic paracentral scotoma in each field, more marked in the left, and consisting of extensions above and below from the blind spot areas.

CASE 10. Mr. TOM B.

February 17, 1904. There are well marked fundus changes. Both optic discs are surrounded with a "honeycomb frame" with some pigment present. The maculae appear involved. In the right eye the changes at the macula almost assume a circinate ring.

R. and L. vision = 6/9.

CASE 11. Mr. FREDERICK B. Died in March, 1934.

January 27, 1904, aet. 38 years. Blacksmith.

R.v. = 6/9.

L.v. = 6/6. There are distinct white spots in both macular areas.

April 4, 1906. R.v. = 6/12 with +1·0 D. sph. = 6/9.

L.v. = 6/12 with +1·0 D.sph. = 6/9.

L. fundus shows spots around the L. macula especially down and in, arranged in a broken circle.

R. fundus shows spots around the R. macula more extensive than in the left.

December 31, 1931. Complains that vision in right eye has become poor for several months.

R.v. = 6/60 with +1·50 D. sph. = 6/24 two letters.

L.v. = 6/12 one letter with +1·0 D. sph. = 6/9 one letter.

R. fundus. At the upper edge of the disc is a small collection of honeycomb spots. In the macular region there is a very gross area of honeycomb choroiditis, and what looks like some very fine haemorrhages in the centre of the area, and also on the lower and inner edge.

L. fundus shows a white hyaline area down and in from the edge of the disc and some more on the outer margin. There is a gross area at the macula but no typical honeycomb appearance.

January 27, 1932. R.v. = 6/60 with +1·50 D. sph. = 6/36.
L.v. with +1·0 D. sph. = 6/9 part.

February 24, 1932 } *In statu quo.*
February 9, 1933 }

March 14, 1934. Died.

CASE 12. Mrs. ROSE J. Widow—no children.

July 10, 1903, aged 45 years.

R.v. = 6/12. L.v. = 6/24 one letter.

Honeycomb choroiditis both eyes.

Notes on writer's examination.

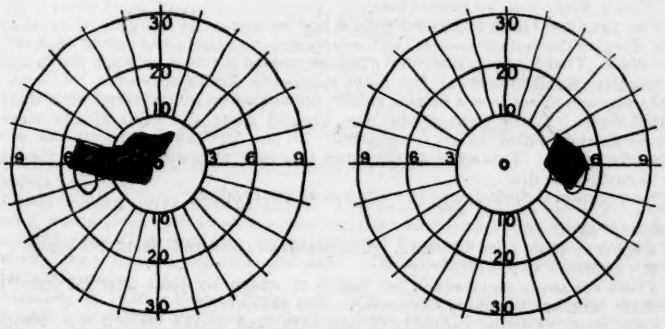
March 4, 1936. Patient is now 78 years.

R.v. = counts fingers at 2 metres.
L.v. = variable P.L. Poor projection. Calcareous opaque lens.
R. lens shows a few peripheral opaque sectors.
Under H. and C. refraction +2·50 / +2·50

R. fundus shows a large area of confluent exudates in the disc and macular region, with very little pigmentation at all. The plaque of confluent exudation shows a central irregular area free from all exudate. There is marked sclerosis of choroidal vessels to be seen in this central free area, and also around the margins of the plaque of exudate, especially below.

CASE 13. MRS. MARIA B.

March 25, 1909, aged 61 years. Complains that sight in right eye has been failing for one year.
R.v. = 6/60 with −1·0 D. sph. = 6/36. R. lens changes are present.
L.v. = 6/12 with +1·0 D. sph. = 6/6 two letters.
The *fundi* show much choroiditis on the outer side of the discs of the honeycomb type with superficial pigmentation.
November 1, 1911. R. lens becoming more opaque, with barely any view of fundus.
July 31, 1912. R.v. = 6/60. L.v. = 6/24 one letter.
There is recent haemorrhage at the left macula.
June 10, 1914. R.v. = 6/60. L.v. = 6/18 with +0·75 D. sph. = 6/12.
August 16, 1916. L v. 6/36 with +2·0 D. sph. = 6/12 part.
April 23, 1919. R.v. = shadow perception. L.v. = 6/60 with gl. 6/12 one letter.
L. fundus shows fine exudates on the outer side of the disc. The honeycomb condition extends to the disc margin. There are some spots on the upper disc margin and on the inner side.
March 12, 1930. *L.* eye shows some peripheral lens changes.
R.v. = P.L. L.v. = appreciation of hand movements.
L. fundus. The area of exudation is now surrounding the disc and sends finger-like processes onto the nasal side.
Notes on writer's examination.
April 18, 1936. Patient is now 88 years.
R. eye. Mature cataract. *L. eye* counts fingers at 1 metre eccentrically.
There is a small lenticular opacity on the nasal side.
L. fundus shows a large confluent area of exudation on both sides of the optic disc with a little scattered pigment.
There is a definite margin to the area of exudation on the nasal side of the disc. On the temporal side the exudates form a yellowish plaque extending between the temporal vessels.



CASE 14. GERTRUDE A. 1° White. January 22, 1936.

CASE 14. Mrs. GERTRUDE A. Widow—no children.

August 15, 1923, aged 30 years. Complains of aching top of head, worse on close work.

R.v. = $\frac{6}{12}$ with $\frac{+0{\cdot}50 \text{ D. sph.}}{-0{\cdot}75 \text{ D. cyl.} \rightarrow 180°}$ v. = $\frac{6}{6}$. Pupils equal and active.

L.v. = 6/5 with $+0{\cdot}50$ D. cyl. $\downarrow 90°$ = 6/5.

Fundi.—Choroidal exudates in both fundi especially around optic discs.

May 21, 1930. R.v. = 6/60. L.v. = 6/9.

Gross central changes in both eyes especially affecting the right macular region.

Blood Sigma for syphilis—Negative

Ordered French iodine minims iii in milk b.d.

August 24, 1932. R.v. = 6/60
L.v. = 6/12 two letters with glasses } Fundi unchanged.

March 7, 1934. L.v. with glasses 6/36 one letter.

L. fundus shows recent additional exudate between disc and macula with small haemorrhage on it.

June 27, 1934. L. fundus unchanged, one old haemorrhage seen.

October 31, 1934. L.v. with glasses = 6/24 two letters.

No haemorrhage seen to-day.

Notes on writer's examinations.

January 22, 1936. R.v. = $\frac{6}{36}$ with $\frac{-1{\cdot}0 \text{ D. sph.}}{+1{\cdot}25 \text{ D. cyl.} \downarrow 115°}$ = $\frac{6}{36}$

L.v. = 6/60. Not improved.

For reading with R. $\frac{+1{\cdot}0 \text{ D. sph.}}{+1{\cdot}25 \text{ D. cyl.} \downarrow 115°}$ and L. $\frac{+1{\cdot}0 \text{ D. sph.}}{+1{\cdot}0 \text{ D. cyl.} \downarrow 90°}$

Binocularly J.12.

R. light sense with George Young's Album = 4th class (1/16).

L. ,, ,, ,, ,, ,, ,, = 5th class (1/8).

Fundi.—No recent haemorrhages are seen.

There are gross confluent exudates in both macular regions, extending between the upper and lower temporal vessels, with coarse pigmentation. There are some discrete spots of exudate on the nasal side of the optic discs especially in the right fundus, and also on the margins of the discs.

April 1, 1936. *Systolic blood pressure* 135 mm. Hg.

June 3, 1936. Condition unchanged. The visual fields are full. The right field shows a paracentral scotoma surrounding the temporal side of the fixation point. The left field shows enlargement of the blind spot.

CASE 15. *New case discovered.* Mr. JOHN B. *With fundus painting.*

April 23, 1936, aged 44 years. Engineer.

R.v. = 6/5 part } Light sense 1st class in both eyes. Colour thresholds, 1st
L.v. = 6/5 full } class in both, but more readily in left eye.

Visual fields full, no scotomata.

The fundi.—There are round colloid bodies along the margins of both optic discs, deep to the retinal vessels, and mostly grouped along the upper edge of the optic discs. There is some scattered irregular coarse pigment between the discs and the maculae, small in amount, but more marked in the right fundus. Around the deeply pigmented spots is a fainter cloudy pigmentation interspersed with discrete hyaloid dots. In the right fundus the hyaloid spots and faint cloudy pigment seem to extend almost up to the macula. In the left fundus the macula is less encroached upon. There are about three tiny pale yellowish spots on the nasal side of each optic disc.

No peripheral disturbance is visible in either fundus.

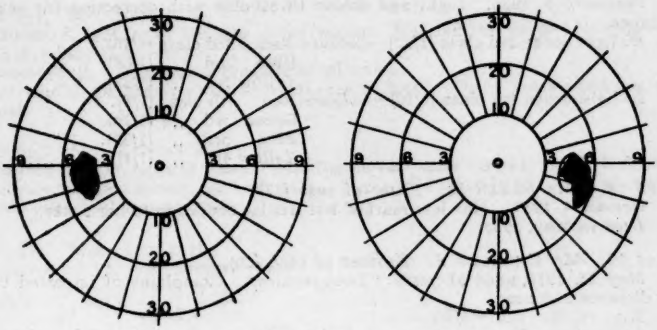
CASE 16. Mrs. JULIA V.

August 5, 1896, aged 46 years. Complains of impaired vision 2–3 years.

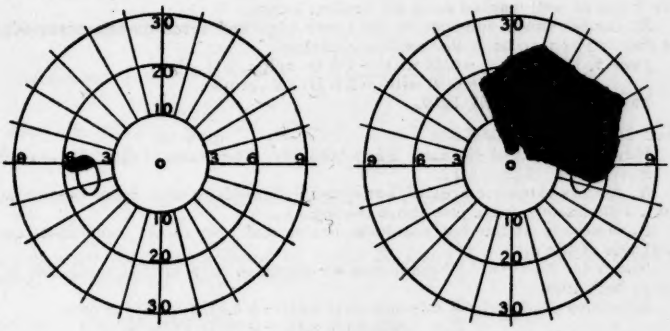
R.v. = 6/60. L.v. = 6/6.

There are seven members in her family of whom six have affected sight, and one sister seen has the same condition as this patient.

Own note.—Fundus paintings of this case seen at the Oxford Eye Hospital show a well developed " honeycomb " picture.



CASE 15. JOHN B. 1° White, April 23, 1936.



CASE 17. LAVINIA N. 1° White. January 8, 1936.

CASE 17. Mrs. LAVINIA N.

Sister of case (18). No other brothers or sisters, and both parents are dead.

October 4, 1933, aet. 50 years. Difficulty in reading for one year. States that vision in left eye has always been poorer than the right.

R.v. = 6/12. L.v. = 6/60.

Both fundi show Doyne's choroiditis.

February 13, 1935. R.v. = 6/36. L.v. = 6/60.

Notes on writer's own examinations.

October 2, 1935. R.v. with $\frac{+1{\cdot}25 \text{ D. sph.}}{+0{\cdot}25 \text{ D. cyl.} \downarrow 70°} = \frac{6}{36}$ one letter.

L.v. with +1·25 D. cyl. $\downarrow 90°$ = 6/36 one letter.

With addition of +1·25 D. sph. J.16 both eyes.

Media are clear.

Fundi.—Deep physiological cups. There are aggregated, discrete, putty-coloured, exudates in both fundi giving a typical honeycomb appearance, especially in the right eye. The exudates are surrounded by shadow rings, with additional scattered, large, coarse, pigment patches. The exudates also extend all round the disc margins.

January 8, 1936. *Visual fields* are full. The right field shows a large pentagonal paracentral scotoma, extending from the blind spot area, above and in close proximity to the fixation point.

February 5, 1936. Light and colour thresholds with correction for reading distance.

R. light sense 3rd class (1/32)—*Colours* Red 3rd class (1/16).
Blue 2nd ,, (1/64).
Yellow 1st ,, (1/32).

L. light sense 4th class (1/10)—*Colours* Red 4th class (1/8).
Green 3rd ,, (1/16).
Blue 3rd ,, (1/32).
Yellow 4th ,, (1/4).

CASE 18. Mrs. SARAH M. Sister of case (17).

October 4, 1933. Has less marked but similar condition to her sister.

Died in May, 1935.

CASE 19. Mr. CHARLES S. Brother of case (20).

May 24, 1916, aged 51 years. Boot repairer. Complains of impaired vision for distance and near.

R.v. = 6/36. L.v. = 6/36.

L. fundus.—There are two round white spots on the inner edge and one on the outer edge of the optic disc; and a rather extensive area of choroidal disturbance with pigmentation in the macular region. The honeycomb condition here is not so well marked as in his brother's case.

R. fundus shows one spot on the inner edge and three on the outer edge of the disc. The macula is also similarly affected.

June 5, 1918. R.v. = 6/36 with −1·0 D. sph. = 6/24.
L.v. = 6/60 with −1·0 D. sph. = 6/60.

Patient died in April, 1919.

CASE 20. Mr. HENRY S.

May 24, 1916, aged 49 years. Farm labourer. Complains of difficulty in reading.

R.v. = 6/24. L.v. = 6/12.

R. fundus extensive area of honeycomb choroiditis with some pigmentation, and a little haemorrhage just above the macula.

L. fundus is similar but not so extensive and also shows some spots on the nasal side of the disc.

November 28, 1917. There is now an extensive area on the nasal side of the disc in both fundi.

November 26, 1919. R.v. = 6/18 part with −1·0 D. sph. = 6/12 part.
L.v. = 6/18 part with −0·75 D. sph. = 6/12.

R. fundus.—The honeycomb area extends about 2 discs breadth to the inner side, and from the superior to the inferior temporal vessels on the outer side; and about 1½ discs breadth beyond the macula.

L. fundus is very nearly as extensive as in the right eye.

March 7, 1924. Condition unchanged.

July 25, 1928. R.v. = 6/60 with −1·0 D. sph.
L.v. = 6/24 with −1·0 D. sph. = 6/18.

Very extensive and typical honeycomb choroiditis in both eyes. The right fundus shows a good deal of pigmentation.

October 22, 1930. R.v. = 6/60. L.v. = 6/60 with −1·0 D. sph. = 6/24.

Gross honeycomb choroiditis.

R. eye very heavily pigmented.

L. eye more atrophic looking.

CASE (A). WILFRED H.

March 8, 1936, aged 13½ years.

R.v. = 6/5. L.v. = 6/5.

Threshold tests, 1st class both eyes for light and colours.

H. and C. instilled.—Nil abnormal seen in fundi. Discs, vessels, and maculae normal.

CASE (B). VERA H.

March 8, 1936, aged 10 years.

R.v. = 6/5. L.v. = 6/5.

Threshold tests, 1st class both eyes for light and colours.

H. and C. instilled.—Nil abnormal seen in fundi. Discs, vessels, and maculae normal.

CASE (C). Mr. GORDON H.
March 8, 1936, aged 31 years. Chairmaker. Married two years. No children.
R.v. = 6/5. L.v. = 6/5.
Fields full. Does not appreciate blind spots.
H. and C. instilled.—Nil abnormal seen in fundi. Discs, vessels and maculae normal.

CASE (D). Mrs. LOUISA MARY E.
March 6, 1936, aged 58 years. Wearing for two years −0·75 D. sphs. R. and L.
R.v. = 6/24 two letters, with gl. = 6/9. L.v. = 6/18 three letters, with gl. = 6/6.
Nil abnormal seen in fundi.

CASE (E). Mrs. ANNIE H.,
September 15, 1920, aged 39 years.

$$\text{R.v.} = \frac{6}{60} \text{ with } \frac{-3{\cdot}25 \text{ D. sph.}}{-1{\cdot}50 \text{ D. cyl.} \downarrow 80^\circ} = \frac{6}{9}$$

$$\text{L.v.} = \frac{6}{60} \text{ with } \frac{-3{\cdot}50 \text{ D. sph.}}{-1{\cdot}25 \text{ D. cyl.} \downarrow} = \frac{6}{9}$$

Notes on writer's examination.

Refraction $\dfrac{-2{\cdot}50}{-5{\cdot}75}$ $\dfrac{-2{\cdot}25}{-4{\cdot}0}$

March 6, 1936. R. with $\dfrac{-4{\cdot}0 \text{ D. sph.}}{-3{\cdot}0 \text{ D. cyl.} \downarrow 100^\circ} = \dfrac{6}{9}$ one letter.

L. with $\dfrac{-3{\cdot}25 \text{ D. sph.}}{-2{\cdot}0 \text{ D. cyl.} \downarrow 90^\circ} = \dfrac{6}{6}$ part

Binocularly 6/5 with addition of +0·25 D. sph. +1·75 D. sphs. added for reading. Nil abnormal seen in fundi.

CASE (F). Mrs. MARTHA W.
March 6, 1936, aged 46 years.
Wearing R. −3·50 D. sph. L. −3·75 D. sph.
R.v. = 6/60 with gl. = 6/6 one letter. L.v. = 6/60 with gl. = 6/6 one letter. Nil abnormal seen in fundi.

CASE (G). Mr. JAMES WILLIAM E.
April 26, 1936, aged 26 years. Gardener.
Wearing glasses for about six years.

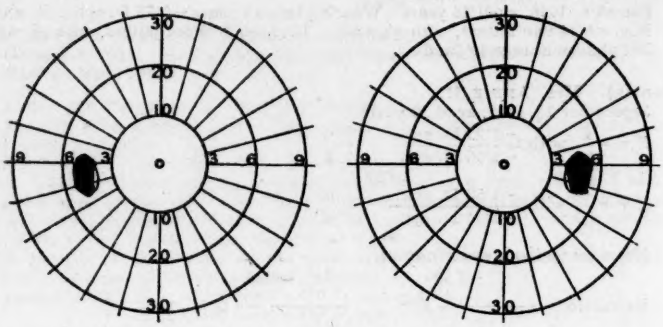
$$\text{R.} \frac{-3{\cdot}25 \text{ D. sph.}}{-1{\cdot}75 \text{ D. cyl.} \rightarrow} \qquad \text{L.} \frac{-3{\cdot}0 \text{ D. sph.}}{-1{\cdot}25 \text{ D. cyl.} \downarrow}$$

R.v. = 6/60 with gl. = 6/6 one letter. L.v. = 6/60 with gl. = 6/6 four letters.
Visual fields full, no scotomata. Nil abnormal seen in fundi.
Light and colour thresholds.
R. with glasses. *Light threshold*, 3rd class. *Colours*, green, 2nd class. Other colours, 1st class.
L. with glasses. *Light threshold*, 3rd class. *Colours*, blue and green, 2nd class. Other colours, 1st class.

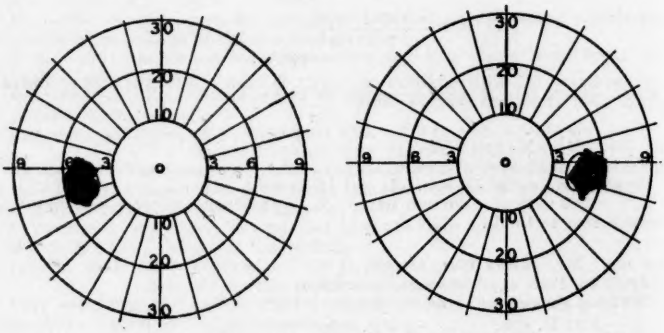
CASE (H). Mr. CEDRIC H.
April 26, 1936, aged 28 years. Clerk.
Wearing glasses since about 10 years of age. Present glasses since November, 1935.

$$\text{R.} \frac{-13{\cdot}0 \text{ D. sph.}}{-3{\cdot}0 \text{ D. cyl.} \rightarrow} \qquad \text{L.} \frac{-13{\cdot}0 \text{ D. sph.}}{-3{\cdot}50 \text{ D. cyl.} \rightarrow}$$

R.v. 6/60, with gl. = 6/9 part. L.v. 6/60, with gl. = 6/9 part.
Fundi show myopic crescents, but nil else abnormal seen.
Visual fields are full, with ? slight enlargement of blind spots.
Threshold tests with glasses.
R. *White*, 2nd class. *Colours*, all 1st class.
L. White and colours, all 1st class.



CASE (*g*). WILLIAM E. 1° White. April 26, 1936.



Case (*h*). CEDRIC H. 1° White. April 26, 1936.

CASE (I). Mr. TERENCE GILBERT W.
January 18, 1933, aged 19 years.
Seen with conjunctivitis and purulent rhinitis.
Nil else recorded.

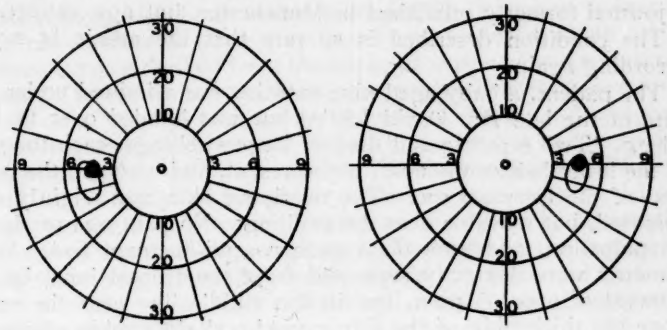
CASE (J). Mr. HARRY B.
April 30, 1936, aged 78 years.
States R. vision failing 9–10 years. L. vision failing 2–3 years.
R.v. = no *P.L.* *L.v.* = *shadow perception.*
Both optic discs show *deep glaucomatous cupping.*
R. eye myopic, with vitreous haze. L. eye shows a few lens opacities.
No choroidal exudates in either eye. *Tension* + in both eyes.
Shallow anterior chambers, pupils slightly dilated and fixed, corneae clear, no pain.

CASE (K). HARRY B.
April 30, 1936, aged 14 years.

R.v. = $\frac{6}{9}$ two letters, with $\frac{-0{\cdot}50 \text{ D. sph.}}{-0{\cdot}50 \text{ D. cyl.} \downarrow 30^\circ} = \frac{6}{5}$

L.v. = $\frac{6}{12}$ with $\frac{-0{\cdot}25 \text{ D. sph.}}{-0{\cdot}25 \text{ D. cyl.} \rightarrow 180^\circ} = \frac{6}{5}$

Thresholds, 1st class for white and colours in both eyes.
Nil abnormal seen in fundi. *Visual fields* full and normal.



CASE (*k*). HARRY B. 1° White. April 30, 1936.

CASE (L). ALAN B.
April 30, 1936, aged 8 years.
Vision 6/5 each eye. Nil abnormal seen in fundi.
Thresholds, 1st class for white and colours in both eyes.

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A CASE OF BILATERAL GANGLIONIC NEUROGLIOMATA OF THE FACE

BY

J. GRAY CLEGG *and* F. CRAVEN MOORE

MANCHESTER

THIS case was reported in March, 1900, in the *Medical Chronicle*, a journal formerly published in Manchester, but now extinct.

The condition described is so rare that the article is worth recording again.

The patient, a baby aged nine months, was admitted under the care of the late Dr. David Little, but was handed over to Dr. Clegg. Two separate and distinct large swellings, each the size of the lower half of the nose, were present, and occupied the position of the lacrymal sac. The overlying skin was slightly discoloured, but movable over the swelling. No pain was produced on palpation, and each mass gave a semi-fluctuant feel. Both tumours were distinctly separated from the frontal bone by an interval of about 5 mm., but in the middle line over the nasal bone the thickening of the skin caused a slight swelling between the tumours. The masses could be moved somewhat laterally and vertically. The conjunctival sacs were both normal, but a small bead of semi-dried muco-purulent matter was always present at the inner canthi. There was no nasal obstruction, the head was of normal shape, and the fontanelles in normal condition for the age of the child, who was perfectly healthy.

At birth a small swelling had been noted on the left side of the nose, and the right swelling appeared a few weeks later. Both gradually increased in size until admission. There had always been a slight discharge at each inner canthus.

The family history was excellent, and the seven other sisters of the patient were all perfectly healthy.

It was assumed that the swellings had some relation to the lacrymal sac, but the tissues seemed too hard to be cut by the Weber's knife, and so a small portion of one tumour was removed for microscopic examination.

Later both tumours were excised; in the right the outer half was distinctly encapsuled, but at the inner side the capsule was adherent to the nasal process of the superior maxilla and to the nasal bone. The left mass was removed through a separate incision, and was found to be smaller and more distinctly encapsuled at the outer side, but here again at the inner side the capsule was adherent to the bone and skin. The wounds healed perfectly.

The child was seen five months later, and was still in perfect health.

The tumours appeared to the naked eye as two ovoid masses, the larger was three quarters of an inch long by half an inch in diameter, and the smaller five-eighths of an inch long by three-eighths of an inch in diameter; they were firm and somewhat elastic in consistency; the surface of each was ragged, of a fleshy appearance, and marked with patches of congestion and haemorrhage. The cut surface presented a uniform grayish pink, somewhat translucent, appearance, and a few small vessels.

Microscopically both tumours presented the same structure; they consisted of a sparsely cellular tissue enclosed within an ill-defined capsule derived from the adjacent muscular and connective tissues. In the former the most conspicuous elements were large ovoid cells, some provided with definite processes, and composed of finely granular protoplasm, in which were one or two large oval vesicular, lightly staining, nuclei. These ganglionic cells were lying in the midst of a peculiar reticulum consisting of delicate fibres, among which were embedded small oval nuclei surrounded by a very scanty cell-protoplasm. Throughout the neuroglial basis there were many thin-walled capillary vessels, and here and there a larger vessel with well-defined walls, coming apparently from the surrounding tissues.

The peripheral parts of the tumours formed, as already mentioned, a kind of ill-defined capsule derived from the surrounding tissues, and served sharply to define the limitations of the growth. Among the fibrous tissue, muscle fibres and blood vessels of which this peripheral zone was composed, there was no indication of any invasion by the tumour tissue.

As regards the nature of the tumours, Professor Delépine expressed as his opinion that they were ganglionic neurogliomata, a view in complete accordance with their minute structure. The existence of the large ganglion-like cells, many with distinct processes embedded in a tissue the peculiarities of which at once indicated its neuroglial nature, and the presence of doubtful nerve fibres seen in a few places, rendered the nature of the tumours beyond doubt. Concerning the pathogenesis of such tumours, it is probable that the majority must be regarded as originating from some part of the central nervous system, generally the brain, and as a result of some developmental disturbance. Axel Key some years ago described a case in which a ganglionic neuroglioma appeared on the left ala nasi of a man aged 31 years; the tumour, which had been noticed for one year only, was about the size of a plum, and distinctly encapsuled. He regarded this case as proving the possibility of the development of a ganglionic tumour from a peripheral nerve independently of preceding ganglion formation. Such a case is, however, we believe unique. The great majority of tumours of this nature which have been described have arisen in direct relation with the brain.

In the present case it seems not improbable, from a consideration of the position of the tumours, and their existence since birth, that they might have arisen in connection with the olfactory lobes, which possibly, as a result of some localised hydrocephalic condition, had protruded through congenital fissures of the skull, and gradually become constricted off with the closure of such defects.

Such a view at least provides the most rational explanation of the various features which have been detailed.

"INFANTILE AND CONGENITAL RETINAL FOLD"*

BY

A. TILLEMA, M.B., Ch.B.(Edin).

HOLLAND

RECENTLY, a number of cases of congenital retinal fold have been noticed. Some of them also showed anterior displacement of the optic papilla into the interior of the globe. Ancona and Kieuwe thought that this condition is caused by persistence of the connecting portion of von Szily's primitive epithelial papilla. Mann suggested that it is due to adhesion of the primary vitreous to the inner layer of the optic cup. Weve considered that the anomaly must arise through contraction of a mesodermal mass of connective tissue and that probably it is hereditary.

None of these writers found signs of inflammation, but Weve observed remnants of haemorrhage in one case. The essential cause of this curious abnormality remained a matter of conjecture and for this reason the following case is interesting.

The parents of a child three-and-a-half years old complained that 5 months before their child had developed a slight divergent squint and that the (right) eye looked strange.

On examination the eye showed no vascular injection, no exudates, the cornea was clear and the iris normal. The lens was turbid in its deep layers, especially at the centre. Behind the lens, temporally and below, a fairly well defined mass was seen against the retina. With dia-scleral illumination no shadow was observed. The intra-ocular tension was +1. Diagnosis uncertain. It was thought best to excise the globe.

Microscopical examination.—The globe measured 22 by 21·5 millimetres. These are normal figures (Fig. 1).

The cornea, anterior chamber and the angle of the anterior chamber were normal. The iris seemed a little more compact

* From the Department of Ophthalmology of the University of Amsterdam. (Professor W. P. C. Zeeman, M.D.).

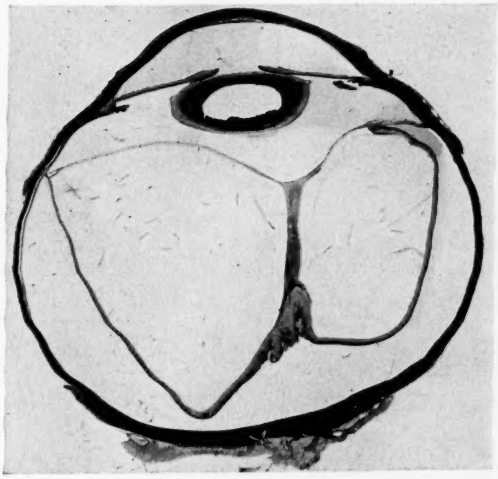

FIG. 1.

Infantile retinal fold. General view.

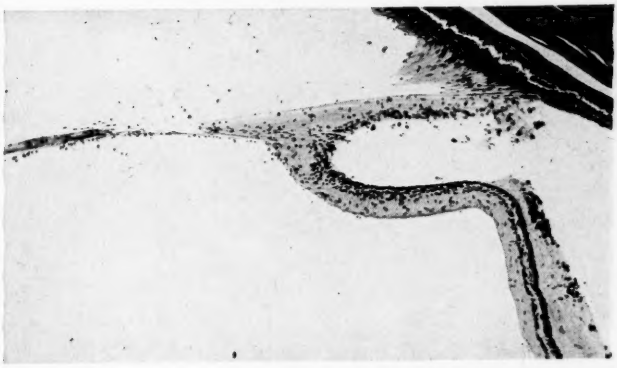

FIG. 2.

Retinal fold with delicate strand of connective tissue.

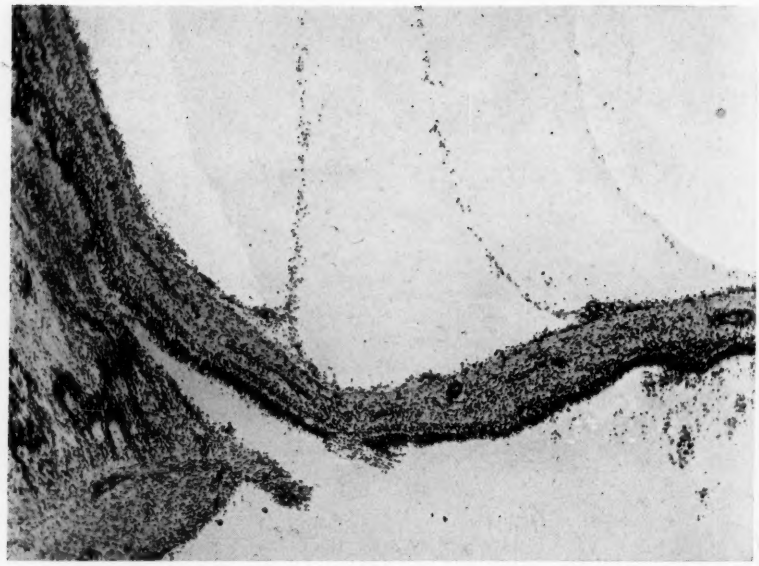

FIG. 3.

Retina showing obvious inflammatory changes.

FIG. 4.

Detachment of choroid by strands of connective tissue. Also note retinal fold.

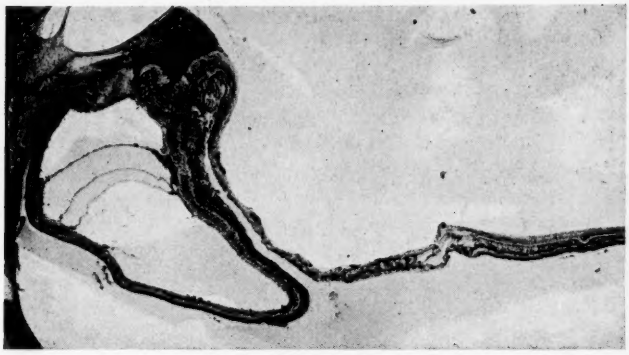

FIG. 5.

Retina showing marked structural changes.

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than usual and its surface seemed smoother, but otherwise no changes could be ascertained. In some places its posterior surface was slightly adherent to the lens near the pupil. The ciliary body and the choroid were quite normal, except in one place temporally and below. This will be described with the retina and vitreous. The lens was not displaced. It was slightly liquefied and vacuolated in the pupillary area of the anterior cortex. The posterior cortex contained very few vacuoles. Delicate fibres of connective tissue were attached to the posterior surface and ran towards the ora serrata.

The retina was completely detached. It showed two folds, one running from the optic papilla towards the pars plana of the ciliary body downwards and slightly outwards (Figs. 1 and 5) and a second just behind the ora serrata running round in a circular direction (Fig. 2). The crests of both folds were attached to strands of connective tissue. The strand adherent to the fold running from the optic papilla (Fig. 5) was thick and ran, like the fold to the ciliary body. The strands adherent to the circular retinal fold behind the ora serrata were delicate and stretched across the globe from one side of the ora serrata towards the other, passing behind and adhering to the lens. All these fibres joined behind the lens in a fairly vascular mass of connective tissue.

One may say that every structure to which these connective tissue strands were attached was displaced into the interior of the globe. The retinal folds have just been mentioned. The optic papilla was found 2 millimetres in front of the sclera, and the optic nerve therefore crossed a subretinal space. The choroid, close to the ora serrata temporally and below, was detached by a strong band of connective tissue joining the central mass (Fig. 4).

The retina showed disturbances of its normal structure. Rods and cones were often missing and the cells of other layers had partly lost their regular arrangement and normal staining properties. Many small spaces were seen filled with a homogeneous coagulum. Scattered leucocytes with segmented nuclei were interspersed between the retinal cells. The retinal veins were surrounded by closely packed lymphocytes. In most cases the vessel wall stained badly while in some cases only remnants of a lumen and of the endothelium were left. Generally speaking the layers of the retina were easily recognised. Close to the large fold, however, the retina suddenly exhibited complete disorganisation and a hole. In this region, below and temporally, the inflammatory changes were most marked (Figs 3 and 5).

The fluid filling the subretinal space contained cellular débris mostly derived from the neural epithelium, also coagulated material and various cells. This coagulated material had been organised here and there to form connective tissue staining red

with van Gieson. Some of the subretinal cells were not easily differentiated because of degeneration, but they were probably derived from the retina. Other cells, however, were undoubtedly leucocytes. This was proved by their finely granular protoplasm, their size as compared to cells inside the blood vessels and by the shape of their (generally segmented) nuclei. The inner surface of the retina was covered with small clusters of similar cells (Fig. 3).

The optic nerve traversed the subretinal space for about 2 millimetres. In its centre a longitudinal tissue-space was seen filled with coagulated fluid. From the optic papilla a strong fibrous band continued forwards (Fig. 1). Its course across the globe towards the ciliary body temporally and below has already been mentioned. This band contained many fibroblasts and cells derived from the retina. It exhibited circumscribed areas of disintegrated tissue (necrosis) and areas densely packed with lymphocytes.

It has already been mentioned that all fibrous membranes and also the large retinal fold joined close to the ora serrata. The fibrous tissue was superficially attached to the choroid which was partly detached (Fig. 4). Although its surface epithelium was disorganised the choroid itself and the pars plana of the ciliary body showed only a slight lymphocytic infiltration, but no inflammatory disturbance. In the angle between the ciliary body and the fibrous bands young delicate connective tissue was seen.

The retina and the connective tissue membranes contained scattered polymorphous leucocytes, especially along their surfaces (Fig. 3).

The central artery divided into several branches close to the optic papilla and was difficult to trace. No remnants of a persistent hyaloid vessel were found.

Discussion

The inflammatory changes were confined almost entirely to the retina and the vitreous body and for this reason it is not very difficult to reconstruct the pathological process. Retinitis, probably metastatic, was followed by vitreous abscess formation. This abscess then subsided and was organised. Fibrous tissue then contracted and pulled every structure to which it was attached towards the centre of the eyeball. Thus the retina, the optic nerve and the choroid were displaced. This could not have happened if the fibrous strands had been attached to the sclera, but it will be remembered that the attachments were superficial everywhere. It will be remembered too that every fold or crest was found to be attached to a band of connective tissue running towards the centre of the eyeball.

Inasmuch as this case closely resembles those described by

Ancona, Mann and Weve it must be decided whether or not its condition represents a stage preceding the condition of the other cases in the literature.

It is quite conceivable that slight and transitory inflammation of the inner layer of the optic cup might arrest normal development. In such cases remnants of inflammation might well be absent. Neither Mann nor Ancona definitely excluded this possibility, but as these writers found no signs of inflammation this problem is left unsolved.

Weve, on the other hand, seriously considered an inflammatory origin as his 8th case seemingly proved this definitely. He then rejected this theory again because the ciliary processes were free while a connective tissue membrane was attached to the pars plana of the ciliary body. A "cyclitic membrane," however, often arises from the pars plana of the ciliary body (*e.g.*, Beets). This case, therefore, was probably caused by cyclitis and the eyeball I described in the previous pages proves Weve's original idea to be correct.

Weve also argued that the majority of his patients were related to each other and such a relationship would render inflammation, of course, very unlikely. This familial occurrence, however, has not been sufficiently proved as only in Weve's 6th and 7th cases is the diagnosis above suspicion.

On the other hand it is very interesting to read that in both Weve's 6th and 7th patients and in the 1st and 4th the condition was observed in both eyes and in the outer and lower quadrant. Moreover, in all these eyes the fundus was pale and three out of these four patients were highly myopic (the refractive error was not mentioned in the first case).

It is tempting, therefore, to suggest a relationship between the high refractive error and the retinal fold, especially as the relationship between myopia and detachment of the retina is so well known in the adult. Moreover, van der Meer, in 9 out of 9 cases of detachment of the retina found a defect of the vitreous body close to the retinal tear. In old cases this defect was covered by a continuous layer of cells. Inasmuch as changes of the vitreous body are common in high myopia (*e.g.*, posterior detachment and synchysis) a lesion of the outer layers of the vitreous body in the very young seems quite conceivable. Regeneration, such as van der Meer found, might be expected to be very active in the very young. In this manner a considerable amount of tissue could be formed which, on contracting, would produce a retinal fold. Such a lesion would have to be close to the ora serrata. It is worth remembering also that this type of retinal fold was observed in the lower temporal quadrant and that "désinsertions" of the retina in the adult are found most frequently in

the same quadrant (Gonin). This theory would also perhaps account for a possible familial occurrence.

As the retinal fold was attached to the optic papilla it could, by exerting only slight traction, prevent the optic papilla from approaching the outer layer of the optic cup. In this way von Szily's primitive optic papilla could persist.

Conclusions

This paper deals with congenital and infantile folds of the retina. In the author's case the anomaly was caused by inflammation, probably retinitis; in Weve's 8th case probably by cyclitis of the pars plana of the ciliary body.

It is suggested that in Weve's 1st, 4th, 6th and 7th patients the condition be related to high myopia.

For all other cases an inhibition of normal development of unknown origin must be accepted; for this group a familial occurrence has not been proved.

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ANNOTATION

Blindness and its Causes

An interesting review of this subject was given at the Annual Conference of the National Society for the Prevention of Blindness at Colombus, Ohio, on December 4, 1936, by Miss C. Edith Kerby, the statistician of the Society. She began by emphasising the importance of an accurate analysis for two reasons. The first is for the sake of the individual, and the second for the sake of those who are planning work for the blind and prevention of blindness. She quotes the case of a man who was reported to an organization

attempting to make a complete register of the blind in one of the States, who had never been certified by an ophthalmologist. It was found that the blindness in this case could be helped by operation and glasses, and the man eventually returned to his former occupation as a sighted individual.

In the United States the Bureau of Census gave the number of blind in 1930 as 65,431. This was admittedly an under estimate; and in the same year the American Foundation for the Blind tried to get at the true figure by comparing census figures with registers kept by Commissions for the blind in certain States, with the resulting figure of 114,000. Miss Kerby says that even this figure is open to question on account of differences in the various States of the concept of who is to be considered blind.

Among 33 States which have blind relief laws there are at the present ten which define blindness in terms of degree of vision, but there are seven degrees, varying from loss of both eyes to visual acuity of 20/200. Fifteen States use indefinite statements such as "vision insufficient for tasks for which eye sight is essential." The remaining States have no definition mentioned in their laws.

There is at present a tendency to set the dividing line between the blind and the seeing at visual acuity of 20/200, and to include in the blind group individuals whose peripheral field is limited to an angular distance no greater than 20 degrees.

The Committee on statistics of the blind would classify the degree of blindness in five groups. (*a*) Totally blind or mere light perception. (*b*) Perception of motion (under 5/200). (*c*) "Travelling sight" (5/200 but beneath 10/200). (*d*) Ability to read large headlines (10/200 but beneath 20/200). (*e*) Border-line group of acuity of 20/200 and those whose actual central acuity may be better than this figure, but in whom there is a field defect.

As to causes of blindness the aetiological factor is summarised in five groups. At the present time the largest group (40 to 60 per cent.) is the "unknown." Next come the infectious diseases, accounting for from 15 to 25 per cent. of cases. Syphilis and ophthalmia neonatorum are the most important members of this group. Accidents come next, 15 per cent. or more of blindness. Congenital and hereditary causes cause about 10 per cent., and lastly non-infectious systemic diseases which account for rather less than 5 per cent.).

The anatomical site in the causes of blindness is also summarised in eight groups. One case in four or thereabouts is due to cataract. Optic atrophy comes next and includes from 15 to 20 per cent. of the cases. Glaucoma causes more than 10 per cent. Choroido-retinal affections account for about 10 per cent. Corneal opacities (largely ophthalmia neonatorum, syphilis and in some places, trachoma) are responsible for about 10 per cent. Developmental anomalies and degenerative changes account for another 10 per

cent. Uveitis (including iris and ciliary body) account for more than 5 per cent., while refractive errors, chiefly myopia, account for a small percentage (under 5 per cent.).

The age at onset of blindness should be stated as definitely as is possible. Miss Kerby gives some interesting figures from the 1920 census, *e.g.*, the incidence of blindness among children under five years of age is about 2 in 10,000. The rate falls during preschool, school and adolescent periods but again reaches 2 at about the age of 35 years. It increases up to the age of 60 when it is about 9 in 10,000; and rapidly thence onwards to 75 in 10,000 at the age of 80 or over. "These figures," she says, "at first glance seem to confirm the popular theory that if one lives long enough, he may become blind. (The chances are one in 130 for persons over 80 years.)"

Miss Kerby ends with a word of warning. Her statistics are based on comparative analysis of such data as are available. The figures are incomplete and too much faith must not be placed in their entire accuracy. It is highly desirable that the statistics which have still to be compiled shall have a much higher degree of uniformity in order that the findings of various studies may be combined and compared. For this reason the recommendations of the Committee on Statistics of the Blind, which set a pattern of minimum standards, are stressed.

ABSTRACTS

I.—CONJUNCTIVA

(1) **McKee, S. Hanford (Montreal).—Two cases of diphtheria of the conjunctiva.** *Canadian Med. Assoc. Jl.*, October, 1936.

(1) **McKee** reports two cases of primary conjunctival diphtheria in a boy aged nine years and a girl aged five years. Each case was of the superficial, or croupous, form.

In the case of the boy, the micro-organism, both morphologically and biochemically, was atypical but guinea-pig inoculation proved it to be a true diphtheria bacillus of mild virulence. In the case of the girl, the degree of virulence was high. In neither case was their evidence of diphtheria in any other region of the body.

The bacteriological report on the case of the boy is worth abstracting. "A guinea-pig was injected with the culture as received. Only a slight local induration resulted. Metachromatic granules were not a prominent feature of the organism, and

its fermentation reactions did not conform to those accepted for the diphtheria bacillus, so that a provisional report " Bacillus Xerosis " was given. On the fourth day the guinea-pig died without obvious lesion and the virulence test was repeated with a larger dose of young culture, giving a control animal 500 units of anti-toxin. After two days the control was perfectly well, while the other animal died in 24 hours with local haemorrhagic oedema and haemorrhagic suprarenals: a typical diphtheria death."

The bacteriologist's note is that in this case " morphologically and biochemically the organism would be missed, but there is no escaping the second guinea-pig test."

R. R. J.

(2) **Thygeson, Philips, and Proctor, Francis I. (Santa Fe, New Mexico).—The filtrability of trachoma virus.** *Arch. of Ophthal.*, June, 1935.

(2) **Thygeson** and **Proctor** selected 14 patients with active chronic trachoma and scraped the anaesthetised conjunctivae (2 per cent butyn) with a platinum spatula. The removed epithelium was then suspended in a few c.c. of sterile nutrient broth and ground lightly in a mortar. Some of this fluid was filtered through a collodion membrane. Subsequent inoculation of the filtered or of the unfiltered material into the conjunctiva of African sphinx-baboons produced the follicular conjunctivitis which is characteristic of trachoma in these animals. The authors feel justified, therefore, in stating that trachoma is due to a filterable virus. The virus occurs in two phases, the large initial body (Lindner) phase and the small elementary body phase. Morphologically it is identical with the virus of inclusion blenorrhoea and is strikingly similar to the virus of psittacosis. The authors are planning experiments on blind human eyes in order to determine whether or not trachomatous infection can be produced by bacterium-free filtrates.

F. A. W-N.

II.—RETINA

(1) **Weve, H. (Utrecht, Netherlands).—Diathermic treatment of giant holes in the retina.** *Arch. of Ophthal.*, August, 1936.

(1) **Weve** finds that the prognosis in cases of retinal detachment with the exception of disinsertion, is worse in cases in which large holes are present, than in those in which there are small

ones. Trauma and the pseudo-disinsertion associated with high myopia may produce such large (" giant ") holes that operation is regarded as hopeless. The author finds that this is not so, and reports some cases of successful surgical intervention. In one of these no less than five operations were performed before the retina went back into position. The author localises the hole by trans-illumination. To effect this, he employs a strong source of light and the indirect method of ophthalmoscopy with a × 20 lens. The operator looks at the retinal hole through the ophthalmoscope and an assistant marks the centre of the illuminated area on the sclera. A ring of surface coagulation at 80° C. is then made along the margin of the hole followed by a ring of perforations with a 1·5 mm. needle electrode of platinum along the inner margin of the ring of surface coagulations. In the central part of the sclera three or four punctures are made with 3·5 mm. electrode while at the actual centre a 4·5 mm. needle is used. The procedure is controlled by repeated ophthalmoscopic examinations and varied according to the exigencies of each case.

F. A. W-N.

(2) **Maggiore (Genoa).—The treatment of retinal detachments by diathermy, with special regard to those cases in which no tear can be seen. (La dia-termo terapia del distacco retinico con particolare riguardo al casi di irreperibile scontinuita della retina).** *Ann. di Ottal.*, June, 1936.

(2) In this interesting paper **Maggiore** gives a short account of the methods of treatment of detachments including those which were employed before Gonin's. He decides in favour of diathermy since by it, many cases, such, for example, as those of disinsertion, can be treated successfully, though they were looked upon by Gonin as impossible. With diathermy it is possible to watch the effect of the operation as it proceeds by means of the ophthalmoscope, and the areas of coagulation may be placed in accordance with the visible signs. When no tear can be found, Gonin did not advise operation; the author has in these cases not hesitated to attempt a cure; he has by extensive diathermy scarring, shut off a large area of the retina; if this did not succeed in re-attaching the retina permanently he has repeated the operation in different regions more than once, and in many cases with complete success. He discusses the contra-indications for operation: multiple rents are unfavourable, not because they cannot be closed but because they show a general degeneration of the retina which makes further rents likely; aphakic eyes, also, often fail; eyes with much reduced tension show that there is severe disturbance of their nutrition, and failure is probable; longstanding detachments which do not reach the macula are not specially unfavourable. The author adds that while we have a very valuable weapon

in diathermy, we must not neglect non-operative treatment entirely, since some cases respond to medical treatment, nor must we forget that some later method of operation may be devised which will enable the surgeon to improve on the present proportion of successes.

HAROLD GRIMSDALE.

(3) **Uyama, Yasuo (Osaka).—On experimental tuberculous periphlebitis retinae with special reference to the immunity of the animal tested. (Zur experimeniellen tuberculösen Periphlebitis retinae mit besonderer Berücksichtigung des Immunitätszustandes des Versuchstieres).** *Arch. f. Ophthal.*, Vol. CXXXV, p. 364.

(3) In this experimental research, carried out on rabbits, **Uyama** sought to discover whether in the production of tuberculous periphlebitis in the retina by the injection of tubercle bacilli into the circulation there was any difference between animals previously treated with repeated subcutaneous injections of tuberculous antigens and animals not so treated.

His results confirmed what had already been found, viz., that in rabbits previously sensitized with tuberculous antigens an early hypersensitivity reaction takes place; in them periphlebitis developed, exclusively in the retina, more frequently than in the control animals, and after much smaller injections of living tubercle bacilli than had been required by previous investigators.

This periphlebitis is primarily and mainly situated in the temporal veins not at the periphery of the retina but between the ora serrata and the optic disc; the vein is not encircled by the infiltration, which invades the perivascular lymph space on the scleral side, or only on one side, of the vein. It consists chiefly of lymphocytes and epithelioid cells (giant cells and caseation being absent). The histological picture is closely analogous to that of periphlebitis in the human eye.

As regards the passage of the tubercle bacilli in the retina it is assumed that when they reach the retinal vessels they pass into, and settle in, the perivascular spaces of the veins through the post-capillary area, or possibly by way of the vitreous.

THOS. SNOWBALL.

CORRESPONDENCE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

GENTLEMEN,—In the November issue of the *Brit. Jl. of Ophthal.*, p. 649, there is a review of the book, Detachment of the Retina, by H. Arruga, translated by R. Castroviejo.

It is very justly stated in this review that there are a number of misspelled words. The book was published shortly after the unfortunate events in Spain started. The author and the translator did not have the opportunity to correct the proofs. The words that are misspelled in the book were properly spelled when the manuscript was submitted to the editors, but it seems that the edition was carried out under unfavourable circumstances by non-English speaking editors.

The author and the translator offer their sincere apologies for a condition beyond their control. Will you be so kind as to publish this letter in the book review section of the *Brit. Jl. of Ophthal.*?

Very sincerely yours,

R. CASTROVIEJO.

635 WEST 165th STREET,
NEW YORK.
December 18, 1936.

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—It is probable that during the time of the Coronation that some old students of Moorfields from Overseas, will be in London. We are, therefore, arranging a short series of demonstrations that they may like to attend, and the following programme is being arranged:—

1. Exhibition of Ophthalmoscopic Conditions, Tuesday, May 4, at 5.30 p.m.
2. Exhibition of Slit-lamp Cases, Thursday, May 6, at 5.30 p.m.
3. Operation Demonstrations: (*a*) Cataract.
   (*b*) Glaucoma.
   (*c*) Detachment.
   (*d*) Squint.
   A selection of the above on the mornings of Monday, May 3, Wednesday, May 5, Friday, May 7.
4. Orthoptics, Tuesday, May 4, at 10 a.m.
5. Pathological exhibitions, Wednesday, May 5, at 5.30 p.m.

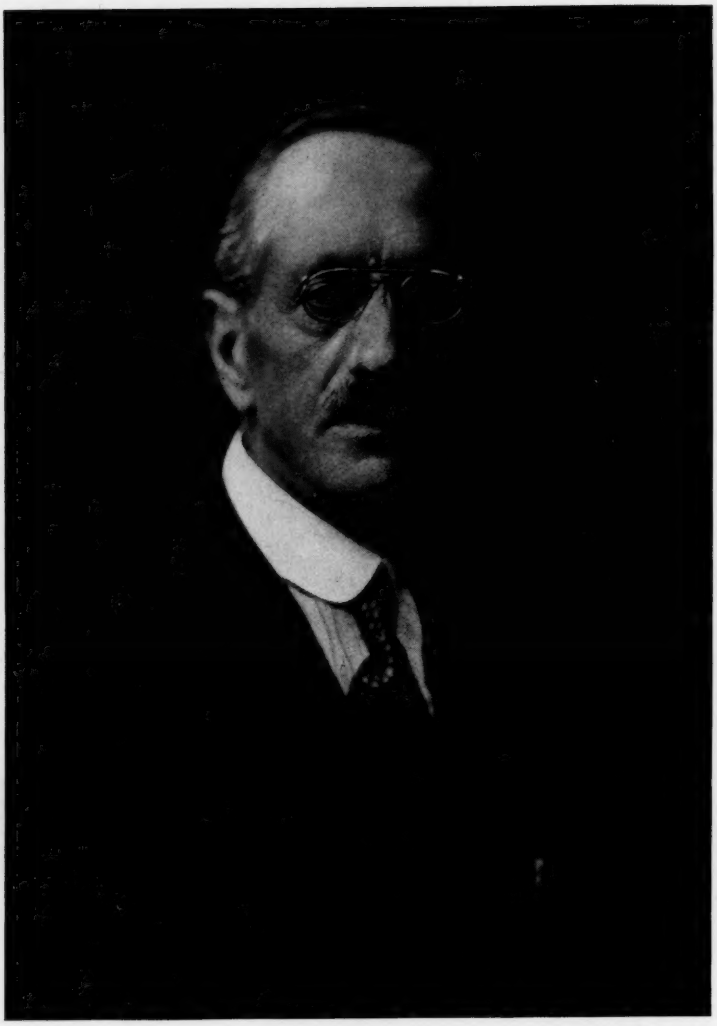

Photo by Davis, Dublin

LOUIS WERNER

UNIV. OF MICH.

We will be pleased to welcome any of our old friends, and it would make arrangements easier if they would be so good as to send us notice of their intention to attend the course.

Yours faithfully,

CHARLES GOULDEN,

Dean of the Medical School,
Royal London Ophthalmic Hospital.

CITY ROAD,
LONDON, E.C.1
January 13, 1937.

OBITUARY

LOUIS WERNER

IRISH Ophthalmology has suffered a severe loss in the death of Louis Werner which occurred in Dublin on November 24 at the age of 78 years.

He was the eldest son of Louis Werner, a French artist, who had studied at the Académie des Beaux-Arts in Paris under Paul Delaroche, and finally settled in Dublin.

At the age of fifteen, the young Louis was sent to the College of La Chapelle near Belfort in Alsace, where he acquired that proficiency in French and German which later proved invaluable to him in his extensive study of contemporary ophthalmic literature.

Returning to Dublin in 1876 he entered Trinity College. At his final Arts examination he obtained the "Large Gold Medal" in Ethics and Metaphysics. In 1880 he entered the Medical School, obtained a "Scholarship," and finally qualified in 1884.

Having decided to devote himself to the practice of ophthalmology, he was appointed to the Staff of the National Eye and Ear Infirmary under Dr. C. E. Fitzgerald and Dr. (later Sir) Henry Swanzy.

During the next few years, Louis Werner played an active part in helping to bring about an amalgamation between this small hospital, established in 1814, and the hospital of St. Mark's, an institution founded in 1844 by Sir William Wilde.

In 1897, he and his colleagues had the gratification of assisting in the establishment of the present Royal Victoria Eye and Ear Hospital. This institution is a worthy monument to the zeal and public spirit displayed by the Irish Ophthalmologists of the nineteenth century with its 134 beds and large annual dispensary attendance.

Louis Werner was best known outside Ireland through his connection with the popular text-book: "Swanzy's Diseases of the

Eye." Between 1907 and 1925 he co-operated with Sir Henry Swanzy in producing a ninth and tenth edition, and on the death of his colleague, he produced three further editions under his own name. The sections in these which deal with optics and neurology are almost solely his own work. A further notable contribution are his beautiful paintings of external diseases and fundus conditions.

Among his many and various communications recorded in the *Transactions*, perhaps the most interesting are a short paper published in 1886, in which he identified the condition "Infiltration vitreuse de la retine" described by Masselon in 1884 with "Central guttate choroiditis": a note on a case of subconjunctival cysticerus (Taenia Solium) published in 1889; and a paper describing a case of "Intra-ocular echinococcus cyst with brood capsules" published in 1903. This last, illustrated by the author's excellent drawings of the macro- and microscopical appearances, together with micro-photographs, has been accepted as the classical description of a condition which is of exceeding rarity in the British Isles.

Louis Werner's reputation in Ireland stood deservedly high. He held many public appointments, including that as Professor of Ophthalmology at University College, Dublin. His enormous private practice was evidence of the confidence that he inspired in the general public.

He had a most retentive memory, and a clear logical mind, characterised by a remarkable rapidity of thought. This rapidity also characterised his operative technique, which was of a very high order. It was a revelation to see him perform a cataract extraction. He had completed the operation, almost before those around had quite realised that he had begun.

He never lost his interest in intellectual pursuits. The fact that he took up the study of Russian, when over sixty years of age, is a proof of unusual mental vitality. His retirement from the "Eye and Ear" in 1934 came at the completion of fifty years of hospital work—surely a notable record of public service. He was a man of simple tastes. He loved music, and was a violinist of no mean order. To see him as the centre of his happy home circle was to see him at his best.

E. M. M.

THOMAS B. HOLLOWAY, M.D.

OPHTHALMOLOGY has suffered a great loss in the death of Dr. Thomas B. Holloway, of Philadelphia, for, besides being an eminent practitioner, he was Professor at the University of Pennsylvania, preparing students for an understanding of the part Ophthalmology bears in medicine, and, as an organizer at the Graduate School connected with the University, he became

identified in the preparation of graduate students for practice in the specialty.

Dr. Holloway had been ill since last December (1935) when he was stricken with pneumonia to which complications were added, so that he could not survive, as was hoped he might, but lingered till August 17, 1936, when he died at his home at Merion, a suburb of Philadelphia, being then sixty-four years of age.

Dr. Holloway was a native of Pennsylvania. After an academic course he entered medical studies at the University, receiving his degree in 1897. After serving an internship in the Philadelphia General Hospital he assisted in various departments of several other hospitals until 1902, when he became associated with the Ophthalmological division at the University Hospital, and from that time onward he devoted himself to that branch, working in company with Dr. de Schweinitz, whose office-assistant he was for a number of years, and with whom he collaborated in several important papers and reports.

He early sought opportunities to serve in important hospital positions, which included the Infirmary for Nervous Diseases, and the Wills Hospital. From 1909 onward he contributed many papers and reports to the proceedings of the numerous societies and fellowships of which he was a member.

On the retirement of Professor de Schweinitz in 1924, Dr. Holloway was elected to succeed him in the Medical School and served as Ophthalmologist to the University Hospital, which position he filled with distinction till his last illness.

His membership included local, State, and National, Medical Associations; the Ophthalmological Society of the United Kingdom and the Société Française d'Ophtalmologie.

For a number of years he was occupied with the affairs of the American Ophthalmological Society: Secretary, Editor, member of Council, and, in 1932, President.

His activities as an Ophthalmologist extended to the School for the Blind at Overbrook: the Society for the Conservation of Vision and that for the Prevention of Blindness; in the Councils of which his advice was sought and followed, as well as it was on the Committee for the International Congress held in Washington in 1922.

His reading was wide in its extent; his memory extraordinarily exact and resourceful, so that in the discussion of cases presented at meetings, Dr. Holloway was always able to add valuable data drawn from his own varied clinical experience.

Possessing a buoyant spirit and ready wit, while at meetings his participation would bring out humerous sallies and such apt quotations that his remarks enlivened many an otherwise dull discussion. As teacher he excelled in his disquisitions during which he presented to the student mind the neurological features of his subjects in a manner strikingly expert and felicitous.

All his days he was interested in field sports; an enthusiastic base-ball player, having played in the College teams. His mid-summer holidays latterly were passed in the Canadian woods.

Dr. Holloway was twice married; a son by the first marriage and his widow survive.

BURTON CHANCE.

NOTES

Death

Mr. ARTHUR LEGGE ROE, the well-known ophthalmic surgeon of Hull, died towards the end of last year at the great age of 82 years. He was trained in Ireland, and took the L.R.C.S.I. in 1873 and joined the Royal Naval Medical Service. On leaving the sea he settled in Hull where he was elected ophthalmic surgeon to the Hull Royal Infirmary. Roe joined the Ophthalmological Society of the United Kingdom in 1898 and kept up his membership till he retired in 1932. He did not write much but a paper of his in the *Brit. Med. Jl.*, 1915, on "Collosol Argentum and its Ophthalmic Uses" led to some correspondence in our columns in Vol. VI. At the time of his death he was consulting surgeon to the Hull Royal Infirmary.

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Ophthalmological Society of the United Kingdom: Annual Congress

THE Annual Congress of the Society will be held in London from April 29 to May 1. There will be a discussion on "The rarer forms of keratitis." Openers will be Mr. F. A. Williamson-Noble (neurological aspects), Mr. Affleck Greeves and Mr. J. H. Doggart (clinical), Mr. Harrison Butler (slit-lamp), Mr. Frank Law (ultra-violet light therapy), and Dr. Douglas Webster (X-ray therapy). It is hoped to hold the Clinical Demonstration at the National Hospital, Queen Square, on May 1. On the afternoon of that day the Management of the *Evening News* has kindly undertaken to show members of the Congress over their printing works at Carmelite House, E.C.1. The Annual Dinner of the Society will be held on April 29.

The Treacher Collins Prize

UNDER the above title the Council of the Ophthalmological Society of the United Kingdom has instituted a prize of £100 awarded triennally for the best essay submitted upon a subject selected by the Council. The prize shall be open to qualified medical practitioners of any nationality. The essay shall be written in the English language. The subject for the first award of the prize is "Cerebro-spinal Disease and its Relation to the Optic Nerve." The closing date for sending in Essays for the first award is

December 31, 1938. Essays should be submitted to the Honorary Secretary, Ophthalmological Society of the United Kingdom, 5, Racquet Court, Fleet Street, E.C.4, from whom also any further particulars can be obtained. No name should be upon any essay, but a distinguishing pseudonym or quotation, which should also be upon a sealed envelope containing the candidate's name and address. This envelope should accompany the essay.

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Congress of Military Medicine and Pharmacy

WE are asked to note that this Congress will be held at Bucharest from June 2 to 10, 1937, and in connection therewith a special tour of Central Europe is being arranged, leaving Paris on May 23. Further details can be obtained from Gellatly, Hankey & Co., Ltd., 62, Pall Mall, S.W.1.

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Royal London Ophthalmic Hospital

THE Annual Dinner of Past and Present Students of the Royal London Ophthalmic Hospital, will be held at the Langham Hotel, Portland Place, on Thursday, March 11, 1937, at 7 for 7.30 p.m. Chairman: T. Harrison Butler, Esq., M.A., D.M., Consulting Surgeon to the Birmingham and Midland Eye Hospital. Secretaries: Charles Goulden, 89, Harley Street, W.1; P. G. Doyne, 69, Queen Anne Street, W.1.

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Orthoptics

AT an examination in Orthoptics held by the Orthoptic Board at the Royal Westminster Ophthalmic Hospital in January, 1937, the following candidates were successful:—Miss K. M. Parsons, Miss F. B. Young, Miss Card, Miss J. Atkinson, Miss B. V. McKenzie, Miss L. B. Jenkins, Miss H. E. Phillips, Miss D. E. Woods, Miss H. G. Lee.

CHARLES LEONARD GIMBLETT,
Chairman.

FUTURE ARRANGEMENTS

1937

February 1.—North of England Ophthalmological Society, at Manchester.

February 2.—Midland Ophthalmological Society, at Derbyshire Royal Infirmary, Derby.

February 5.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

February 12.—Royal Society of Medicine; Section of Ophthalmology.

March 5.—North of England Ophthalmological Society, at Liverpool.

March 12.—Royal Society of Medicine; Section of Ophthalmology (Clinical Meeting).

April 9.—North of England Ophthalmological Society, at Sheffield.

April 16.—Midland Ophthalmological Society, at the Nottingham Eye Infirmary.

April 29, 30 *and May* 1.—The Annual Congress of the Ophthalmological Society of the United Kingdom to be held at the Royal Society of Medicine, 1, Wimpole Street, London, W.1.

June 4.—Midland Ophthalmological Society, at Shrewsbury Eye Hospital.

June 11.—Royal Society of Medicine; Section of Ophthalmology (Annual).

July 8-10.—Oxford Ophthalmological Congress, at Oxford.

October 1.—Midland Ophthalmological Society, at the Eye Hospital, Birmingham (Annual Meeting).

December 7.—Midland Ophthalmological Society, at Birmingham Eye Hospital (Middlemore Lecture).

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *December, 1936.*

CARROLL. "Alcohol" amblyopia, pellagra, polyneuritis. Report of 10 cases.

BALLANTYNE. The Tennent Chair and the Tennent Memorial Institute of Ophthalmology, Glasgow.

GUMP, HESTER and LOHR. Monocytic chloroma. (Reticulocystoblastoma with monocytic leukemia).

BRUCK. Deposits of fat in trachomatous pannus.

THOMSON and CAVE. Experimental detachment of the retina; treatment with subretinal injections of blood plasma.

SCALA and SPIEGEL. Cortical innervation of ocular movements.

MARLOW. Tests for heterophoria: a protest.

KRAUSE. Carnosine of the ocular tissues.

GLICK and BISKIND. Studies in histochemistry: X. Distribution of vitamin C in the lens of the eye.

JAMESON. Operative treatment of detached retina: principles observed by six individual operators.

BELLOWS and ROSNER. Biochemistry of the lens: X. Preparation of glutathione from the crystalline lens.

TAN. Formation of new vessels in the vitreous.

American Journal of Ophthalmology. *December, 1936.*

WETZEL. Aneurysm of the internal carotid artery with atrophy and compression of the optic nerve.

BERENS, NILSON and CHAPMAN. Iritis produced in rabbits' eyes by the intravenous injection of crude and purified cultures of bacteria isolated from patients with certain inflammatory eye diseases.

CARROLL and FRANKLIN. Tobacco amblyopia; alcohol amblyopia.

BOTHMAN and BLAESS. Results of the surgery of glaucoma.

MARSHALL and LAIRD. Ocular changes in multiple sclerosis.

ABRAHAM. Glaucoma in amblyopia.

HARDY. The rôle of paracentesis in ophthalmology.

ROSENBAUM. Unilateral congenital anophthalmos with orbito-palpebral cyst.

Archives d'Ophtalmologie. *December, 1936.*

BONNET, PAUFIQUE and SARRAZIN. Recurrent retino-vitreous haemorrhages in young subjects and their possible relation to tuberculosis.

HALBRON. Ophthalmological sequelae to the radical cure of fronto-ethmoidal sinusitis.

ARGAUD and COUADAU. General considerations on psammomata, in particular a cystopsammoma of the papilla.

PAULA-SANTOS. Traumatic hernia of the vitreous body in the form of a sac in the anterior chamber and considerations on the limiting of the vitreous.

POLEFF. Culture of "Rickettsias du trachome" in vitro.

COULOUMA and LESENNE. Cure of a central retinal vein thrombosis following a dental extraction.

Annales d'Oculistique. *November, 1936.*

SÉDAN and JAYLE. Considerations on central retinal artery spasms.

BUJADOUX. Spherical perimeter. Modification of the classical perimeter.

PUSCARIU and LAZARESCU. The eye and general infections. Conjunctivitis and gonococcal arthritis.

Klinische Monatsblätter für Augenheilkunde. *November, 1936.*

STOCK. Hitherto undescribed rapidly progressive form of retinal pigment degeneration with total blindness.

TERTSCH. Can it be established as a law that retinal pigment degeneration is hereditary in families of the same blood group?

DENECKE. The prognosis of uveosarcoma.

BASSIN. Vitreous detachment.

SCHEERER. Constitution and the fundus.

LÖWENSTEIN. A hitherto undescribed manifestation in the vitreous associated with congenital excavations of the optic papilla.

KAMOGAWA. Blood pressure alterations in the central retinal artery through postural changes of the convalescent patient.

LAZARESCU and DAMIAN. Kalzana and calcium therapy for eczematous blepharo-kerato-conjunctivitis.

STREBEL. Allergic lid oedema and its cause; Tropho-allergic conjunctivitis with lid oedema. One sided upper lid oedema from tar idiosyncrasy. Lower lid oedema from acute pollen allergy.

KESSLER. The trichromatic system in the service of the dichromatous.

Archivio di Ottalmologia. *November, 1936.*

ROSSI. The clinical features of some ocular syndromes of hepatic origin.

PORTCHESE. Oculo-cardiac reflex and glyco-regulation.

BARATTA. The behaviour of the intra-ocular pressure after paracentesis of the anterior chamber in eyes with normal tension, in chronic simple glaucoma and in anterior uveitis with hypotension.

Annali di Ottalmologia e Clinica Oculistica. *July, 1936.*

CONTINO. Anterior haemorrhagic glaucoma.
SANTONASTASO. The pathogenesis of buphthalmos.
CAMPOS. Research on retinal compensation.

Bollettino d'Oculistica. *October, 1936.*

MOTOLESE. Two interesting observations on panophthalmitis and traumatic uveitis of rapid development.
RINALDI and ARSLAN. Exudative retinitis following sphenoidal sinusitis with hyperostosis.
SPINELLI. Treatment of retinal detachment with diathermo-coagulation.
NASTRI. Research on the physiopathology of the subject of an ophthalmic operation.

November, 1936.

CATTANEO. Hypertensive iridocyclitis.
SCHUPFER. Acute retrobulbar neuritis with particular regard to its cause, pathogenesis and diagnosis.
DENTI, Fundus lesions from serious thoracic injuries.
BOSSALINO. Contribution to the study of filamentous keratitis.
RINDELLO. Research on lysozyme in relation to some interesting ophthalmological questions.

Revista Oto-Neuro-Oftalmologica. *October, 1936.*

BISTIS. Retinal and general hypertension. Ocular disturbance.
VICTORIA and PAVIA. The adiposo-genitalis syndrome and retinal alterations.

November, 1936.

PAVIA. Infra-red ophthalmoscopy and retinography.
MENDOZA. Treatment of alternating strabismus with equal retinal images.
PAVIA. Cataract. Two models of hand suction apparatus for extraction.

Revista Oto-Neuro-Oftalmologica y de Cirugia Neurologica.

January, 1937.

ALMEIDA and SALAS. Epibulbar tumours.
PAVIA and DIEZ. Vitamins A and C in ophthalmology.
MERCANDINO. Colours which promote ocular comfort in an operating theatre.

Archivos Sud-Americanos de Oftalmologia. *September, 1936.*

CHARLIN. Hemerolopia from avitaminosis.
LUQUE. Alterations of the fundus in Nicolas Favre's lymphogranulomatosis.
LENNON. Skeleton free ocular radiographs.